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POST-OPERATIVE COMPLICATIONS.[1]

By W. H. NEILD,
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ONLY certain aspects of this vast subject can be discussed, and the topics chosen either are of particular importance or represent complications of which I have had experience.

Pyogenic Wound Infection.

The high incidence of wound sepsis, largely due to the *Staphylococcus aureus*, is causing the very greatest concern to both surgeons and hospital administrators alike. In a recent publication from the Royal Melbourne Hospital, it was disclosed that no less than 18% of operative wounds became infected and 5% of these infections were serious. Statistics from The Royal Newcastle Hospital for 1958 revealed that in one in three of all emergency cases and in about one in seven of the elective cases wound sepsis developed. In the latter figure there was no differentiation between mild and severe infection. Obviously these figures give no grounds whatever for complacency. Wound infection prolongs the period in hospital, increases suffering and, if it is severe, predisposes to ventral herniation. Occasionally wound sepsis is so serious that it threatens life, by producing fulminating cellulitis and septicæmia.

It is important to realize that in most cases infection is introduced into the wound at the time of operation, that the surgical team represents the greatest potential source of infection to the patient, and that lengthy procedures are much more likely to be attended by septic wounds than short ones. The only answer to this problem so far is the strictest adherence to the minutiæ of the aseptic technique.

Post-Operative Gas Gangrene.

This disastrous form of wound infection occurs occasionally after amputation of the inferior extremity for ischæmic gangrene. Macey and Bickel in 1942 reported an incidence of this complication of 4% in 264 amputations, and located the organisms in the perivascular lymphatics. A case of my own, as well as two others in The Royal Newcastle Hospital, all proved rapidly fatal. Gas gangrene antitoxic serum has been regularly used for prophylaxis, but the series is relatively small and uncontrolled.

Post-Operative Tetanus.

Recently in Newcastle a bone-grafting operation was undertaken in a case in which non-union had resulted from a compound fracture 18 months before. Ten days after the elective bone-grafting procedure the patient developed tetanus. This patient eventually survived, after an extremely stormy and dangerous illness.

The complication was well known after World War I, the tetanus spores apparently being present in the wound, and the secondary corrective orthopædic procedure was responsible for promoting conditions favourable to the growth of the tetanus spores. After World War II,

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 25, 1959.

patients were operated on who had been actively immunized against tetanus. As grave doubts exist about the efficacy of anti-tetanus serum, it would appear wise to induce active immunization against tetanus for this type of case.

Burst Abdomen.

Burst abdomen is one of the most dramatic post-operative complications, and commonly takes place between the tenth and fourteenth days. In my own experience, sepsis has not been one of the serious predisposing factors. The role of hypoproteinæmia and of vitamin C deficiency in impairing wound healing is well known, and these defects should be corrected before elective surgery is undertaken.

The majority of cases occur in the middle-aged or elderly. Cancer sufferers appear to be especially liable to wound dehiscence, presumably because of their impaired nutrition. Paramedian and mid-line wounds, especially in the upper part of the abdomen, are more likely to produce a burst abdomen than muscle-splitting incisions. Coughing and vomiting, by raising intraabdominal pressure, may cause the wound to give way.

One of the best-known and best-documented papers on this subject came from Wolfe in 1950, who published the results from the Veterans Administration Hospital, New York. He recorded the results in 1700 consecutive laparotomies, and found that the incidence was 2·6%, with a mortality of 11%. There were many ventral herniæ in patients who survived.

The most important factor in prophylaxis is careful and meticulous closure of the anterior sheath of the rectus with interrupted sutures. Chromicized catgut is most commonly used; but non-absorbable suture material, were it not for the danger of persistent discharging sinuses, would obviously be preferable. After the strong claims put forward for stainless steel wire by Lawrence Abel and Rose, I adopted this suture material. A continuous suture was employed. Subsequent X-ray examination of some of these patients showed that the steel wire fragmented at the point where it was kinked, and its use was abandoned.

Tension sutures are thought to be not only useless, but dangerous. Deep dehiscence of the wound and impingement of the bowel on the tension sutures strung across the wound may produce a fistula.

Post-Operative Hæmorrhage.

It is the aim of all surgeons to establish meticulous hæmostasis at the time of the operation, and most times this can be successfully achieved; but occasionally reactionary post-operative hæmorrhage is a cause for real concern. Three particular circumstances come to mind:

1. Bleeding from the suture line after partial gastrectomy. This can be averted by making the final suture in the posterior layer of the anastomosis a continuous locking stitch and using three layers anteriorly. Prior to commencing the anterior layers, the stump of the stomach, if held in a clamp, should be released and any bleeding points carefully underrun.

2. Bleeding from the perineal wound after excision of the rectum by synchronous combined abdomino-perineal resection. Should the perineal operator close his wound before the pre-operative blood level has been restored, serious reactionary hæmorrhage may occur after the patient has left the operating theatre.

3. Bleeding after thyroidectomy. Here bleeding, by causing tracheal compression, may seriously threaten life. I have had three cases in some 200 thyroidectomies. The essential treatment in such cases is to restore the airway by tracheotomy. The dictum of the late F. H. Lahey should be borne in mind. He taught that the time to perform tracheotomy for this condition was when the surgeon was beginning to wonder whether it was necessary.

Paralytic Ileus.

For twenty years now the treatment of paralytic ileus has been standardized. It consists in gastric or intestinal suction and in the maintenance of the fluid and electrolyte balance by intravenous therapy.

It is my view that for paralytic ileus or plastic peritonitis a Wangensteen tube passed via the nose into the stomach is just as effective as the Miller-Abbott tube or its numerous modifications. This, of course, does not apply to organic obstruction of the small intestine due to long-standing adhesions. However, it is in just this class of case that the greatest difficulty is encountered in establishing intestinal intubation. The Grafton-Smith tube so warmly advocated by Wangensteen would almost certainly lead to a greater number of successes.

Post-Operative Choleperitoneum.

Post-operative choleperitoneum, I believe, is rather commoner than is imagined. Within three years of graduation I saw three cases; all the patients passed rapidly into a state of peripheral circulatory failure, none of the abdomens were reopened, and all three died within 72 hours. All surgeons performing operations on the gall-bladder and biliary tract should be interested in and familiar with this condition. Graham McKenzie, of the Royal Melbourne Hospital, published an admirable paper on the subject in 1955. The cases fall into two categories, acute and subacute. In the acute cases, the outstanding and dominating feature is shock. The patient is vaguely unwell, complains of discomfort rather than actual pain in the abdomen and passes into a state of shock within 48 to 72 hours. Of the 20 cases described by McKenzie, seven fell into this category and there were five deaths. In the subacute type there is a latent interval of days or even weeks before the symptoms become manifest. The onset is commonly with pain, which subsides after an hour or two only to recur; but the pain is usually of sudden onset and is located either in the right hypochondrium or in the epigastrium. Other clinical features are vomiting, abdominal distension, mild jaundice and persistent tachycardia.

It is well to keep in mind the pithy aphorism: "The treatment of choleperitoneum is prevention." This is worthy advice; but not all cases are preventible. However, careful ligation of the cystic duct with linen or silk, the accurate closure of the incision in the common duct and oversewing of the gall-bladder bed will help to reduce the incidence of the condition, but will not entirely eliminate it. It is important to keep the condition in mind, as thereby some cases which would probably go on to a fatal termination will end in recovery.

The treatment is, in the acute cases, prompt reopening of the abdomen. Here the diagnosis lies between reactionary hæmorrhage and choleperitoneum, and in either event reopening of the abdomen is indicated. As a rule the treatment consists in evacuation of the bile and the institution of drainage in the subhepatic pouch. It is usually difficult to ascertain the source of the biliary leak. This was so in 12 out of the 20 cases recorded by McKenzie. In the subacute cases, which run a more leisurely course, the evacuation of the bile is, of course, essential, but the great majority of these patients survive. There were only two deaths in McKenzie's cases, and these were cases in which the abdomen was not reopened. In my own experience of about some 500 cholecystectomies, I have had five cases; four of the patients survived with reopening of the abdomen.

Thrombo-Embolic Disease.

The importance of thrombo-embolic disease lies in the threat to life from pulmonary embolus, and in the lifelong disability which is apt to follow a "white leg" in the affected limb. As is well known, in most of these cases the origin is in the deep veins of the calf—i.e., in the large venous sinuses of the soleus. The condition is first one of phlebo-thrombosis, which is silent and dangerous; it is quite asymptomatic in the first instance, pain in the calf occurring only two or three days afterwards. Fatal pulmonary embolus is at least five to ten times greater. The incidence of thrombosis is hard to determine, as it will vary according to the fervour with which the condition is looked for. At least half the fatal cases of

pulmonary embolus occur without symptoms in the calf, as well as many of the non-fatal ones. The signs of a deep vein thrombosis are well known, and they constitute pulmonary embolus as well as the local signs in the leg, i.e. the tender calf, the presence of Homan's sign, tenderness and œdema over the inner aspect of the ankle, and finally unexplained low-grade pyrexia.

Prophylaxis of the condition is most important and will help in reducing its incidence. Prophylactic measures are as follows. (i) The calves of the unconscious patient should be protected. For instance, (*a*) Wangensteen recommends binding the legs of all operation patients firmly with crêpe bandages, (*b*) Hamilton Bailey advocates the elevation of the legs so that the heels rest on rubber sponges and the calves do not impinge on the operating table. This may save damage to the intima. (ii) Deep breathing and leg exercises should be taught preferably before operation, and should begin as soon as the patient is conscious. (iii) The foot of the bed should be raised nine inches while the patient is unconscious and lying flat. This is the application of work by Paling Wright, which does not seem to have received adequate recognition. By means of radio-active sodium she has shown that the venous return from the inferior extremities is accelerated four times if the inferior extremities are raised above the level of the auricles. (iv) Early ambulation should be practised. By early ambulation is meant that the patient must get out of bed and walk. It is utterly futile to have the patient out of bed sitting in a chair alongside the bed. This is not early ambulation, and gives the patient no protection whatever from thrombo-embolic disease. The patient is certainly better off lying in bed provided he moves his legs.

The treatment of the condition is as follows. (i) When massive overwhelming pulmonary embolus occurs, sudden death supervenes and no treatment is practicable. (ii) Cases in which gross pulmonary embolus has taken place and life hangs by a thread for an hour or two would appear to justify a revival of interest in the Trendelenburg operation. (iii) In non-fatal pulmonary embolus treatment is a matter of urgency. It consists in raising the foot of the bed as previously mentioned and the administration of heparin. Heparin is given through a Gordh's needle, and 5000 units are administered every four hours for four days. At the end of this time the clot will have become adherent to the vein wall, and the prospects of its propagation are not high.

Obviously the best results will be obtained where surgeons and nursing staff alike are conscious of this condition and are looking for it. A complaint of pain in the calf should be sought for with the daily visit, and the calves should be examined. The calves are bandaged on suspicion, and it is as well to administer heparin. I have not cared to give heparin before the fifth day. Heparin should on no account be given subcutaneously or intramuscularly, as the injections are painful and large hæmatomas may occur.

As for the dicoumarol type drugs, I have abandoned their use completely. They are unreliable in absorption and effect, and I have had a patient under my care who, whilst under treatment with "Dindevan", developed a thrombus in the other leg. It is well to realize that the condition is commonly bilateral, and whilst fairly advanced in one leg is asymptomatic in the other; in consequence, if firm bandaging is undertaken, both legs should be bandaged.

The sequelæ in the leg are very serious in some instances, as the condition may pass on to thrombo-phlebitis, producing the familiar white leg. In at least three-quarters of these cases there is serious subsequent trouble. This consists in a heavy œdematous leg, a painful leg and induration of the skin, which may go on to ulceration. These patients are treated by firm bandaging and elevation of the limb to promote the venous return.

Patients with phlegmasia alba dolens, after they leave hospital, are usually advised to wear some type of external support, such as a stocking or firm crêpe bandage. They should be watched, as subsequently the veins recanalize and the deep perforating veins become patent.

Induration in the gaiter area means that the perforating veins of Cockett have become incompetent, and the advisability of tying them off should be considered before irreversible changes have taken place in the skin.

Conclusion.

It is regretted that no further post-operative complications can be discussed. The subject is one of tremendous proportions, and it is not possible in the time and space available to touch on more than a few topics.

Reference.

McKENZIE, G. (1954-1955), "Extravasation of Bile after Operations on the Biliary Tract", *Aust. N.Z. J. Surg.*, 24: 181.

POST-OPERATIVE COMPLICATIONS.[1]

By ANTHONY S. PATON, F.F.A.R.C.S. (England),
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POST-OPERATIVE NAUSEA AND VOMITING.

I REFER here to nausea and vomiting occurring after operation and due to causes other than those directly attributable to any pathological condition affecting the patient.

You may well ask at this stage: "Is this condition of sufficient importance, in this ever more rapidly developing electronic age, to merit discussion at a learned gathering such as this?" Personally, I think so, and I am sure that any one of you present who has suffered this distressing complaint will readily agree with me. As medicine becomes more and more specialized and scientific with each passing day, there is unfortunately a growing tendency to overlook the simpler things in life and in the practice of medicine. Post-operative nausea and vomiting is one of these simple things which, when it does occur, is only too often readily accepted as an inevitable complication of anæsthesia.

Perhaps this is not unreasonable, when one considers that from 1846 to 1934 the only satisfactory anæsthetic agents available were those that could be administered by inhalational methods, and all of these possess the property of causing nausea and vomiting. It is not difficult to see how, over this period of 98 years, post-operative nausea and vomiting came to be regarded more or less as a *sine qua non* of anæsthesia.

However, with the introduction of "Pentothal Sodium" by Lundy in 1934, people began to realize that it was possible to administer an anæsthetic without its being followed by nausea and vomiting. Then, with the introduction of relaxants in 1944 and the consequent less widespread use of some of the inhalational anæsthetic agents, the incidence of post-operative nausea and vomiting dropped even further. However, it is still a common enough complication of anæsthesia for us to review some of the ætiological factors and to consider some of the factors whereby we can minimze its occurrence and lessen its severity.

Incidence.

How often does post-operative nausea and vomiting occur? Many authors have published figures of the incidence of post-operative vomiting, as follows:

Waters (1936), 40% (10,000 patients).		
Davies (1941), 44% (1000 patients).		
Rubin and Metz-Rubin	(1951)	27% to 75%.
Albert and Cookley	(1954)	
Dent *et alii*	(1955)	
Knapp and Beecher	(1956)	

The large variation in these figures is due to the large number of factors which may cause post-operative vomiting.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 25, 1959.

Ætiological Factors.

Type of Patient.

Human beings, broadly speaking, can be divided into two groups: (*a*) vomiters, (*b*) non-vomiters. In the first group are those unfortunate people subject to motion-sickness, those who vomit after simple dietary indiscretions, those subject to bilious attacks, and those women who are unable to survive the first three months of pregnancy without a daily bout of morning sickness. In the second group are those hardy individuals who go gaily through life without ever having to avail themselves of this physiological, if undignified, means of emptying the stomach. Most of the patients who suffer from post-operative vomiting will fall into the first group.

Age of Patient.

The age of the patient is not of great significance as a causal factor. Post-operative vomiting is certainly more common in children than in adults; but this is due more to the type of premedication and anæsthetic given to children than to their age. However, the incidence of vomiting does decrease with advancing years, and is probably maximal in the 20 to 40 years age group.

Sex.

Post-operative vomiting is more common in women than in men. It occurs about twice as frequently in women as in men.

Condition of Stomach.

Vomiting is obviously more likely to occur post-operatively if the stomach is not emptied prior to operation. This is part of routine preparation for operation; but gastric emptying may be delayed for a number of reasons. Anxiety, trauma, premedication and labour will all delay, and may even prevent, gastric emptying owing to delay or arrest of gastric function. With regard to trauma, it is important to realize that the significant times are the time of the operation in relation to the time of the accident rather than to the time of the last meal. A patient who has suffered trauma always has a full stomach, regardless of how long one may delay operation.

Swallowing of ether vapour and blood will also cause vomiting, even though the stomach is empty prior to operation.

Type of Anæsthetic Agent.

Of the inhalational anæsthetic agents commonly used, ether always has been and still remains the most potent agent in causing post-operative vomiting (33%). Of the others, cyclopropane and "Trilene" share about equal blame (25%), while nitrous oxide and oxygen is associated with the lowest vomiting rate, which is still significantly high (15% to 20%). When we come to the intravenous agents, we find that thiopentone and the relaxants do not significantly affect the incidence, but pethidine given intravenously does, and may even double the post-operative vomiting rate.

Type of Operation.

Three factors influence the incidence of post-operative vomiting as regards the operation. The first is the site. Post-operative vomiting is more common after abdominal operations, particularly when there has been much handling of and traction on stomach and gut. The incidence of vomiting after fenestration is high (75%), and also, strangely enough, after dilatation and curettage of the uterus which, although a short operation, is nevertheless a very stimulating one—and also, of course, 100% of the subjects are women. The other factors are the duration and severity of the procedure. The longer the operation and the more severe its nature, the more common will vomiting become.

Premedication and Post-Operative Medication.

The drugs used for premedication and post-operative medication are probably the greatest single cause of post-operative vomiting. I suppose that the opiates and opiate derivatives are the most commonly used premedicant drugs, presumably because they give the most satisfactory results. But at the same time these drugs are the most potent causes of vomiting.

If we briefly consider the pharmacology of these drugs, we are not left for long wondering why this should be. Let us take morphine as an example.

Morphine in the human exerts its action at some 10 to 12 different points, mainly in the central nervous system, but these may be summarized as two principal actions: (*a*) a depressant action (which we may call the good effect), (*b*) a stimulant action (which we may call the bad effect). By administering morphine to patients, we take advantage of the depressant action to achieve analgesia, sedation, euphoria and depression of certain reflexes such as the cough reflex. Simultaneously, however, the patient must suffer the disadvantages of the stimulant action, of which nausea and vomiting are two. Morphine causes vomiting by acting on the chemoreceptor trigger zone situated in the lateral reticular formation, and when this trigger zone receives sufficient stimuli, these overflow and stimulate the vomiting centre. Further, the effect of morphine on stomach and small intestine is to increase tone and decrease activity. This peripheral effect will summate with the central effect on the trigger zone. One-third of normal subjects experience nausea, while one-tenth of normal subjects will vomit after the administration of morphine.

Drug Idiosyncrasy.

By idiosyncrasy we mean an abnormal response by a patient to an otherwise normal dose of a drug; or, to put it another way, the side-effects of the drug become the main action of the drug, while the main action of the drug becomes insignificant. This is seen fairly commonly with morphine and not so commonly with pethidine. However, when patients do show an idiosyncrasy to pethidine, the effects are quite often severe.

Prevention

The incidence of post-operative nausea and vomiting can be reduced. The incidence of nausea quoted in a recent series of 2500 cases was 35% and that of vomiting 28%, which is in pretty close agreement with those of earlier figures. These figures may surprise you, as they did me. There are a certain number of people who will vomit after operation regardless of all considerations, and these people would constitute about 8% to 10% of those being subjected to operation. This leaves us with about 20%—and I think that, with a little more care and attention to detail, we could eliminate that 20%.

Anticipation.

It is usually possible to obtain from the patient's history, age and sex, and an evaluation of the factors mentioned before, a reasonably accurate estimation of the likelihood of post-operative vomiting. Having done this, we can then take steps to minimize this likelihood.

Premedication.

In the majority of cases, premedication is given unthinkingly and in too high a dosage. What are the objects of premedication? They are: (i) to achieve sedation by allaying anxiety and apprehension; (ii) to produce some degree of analgesia and so aid the onset of anæsthesia; (iii) to depress reflex activity. Now we can do this, either by depressing the central nervous system sufficiently, or by altering its activity in some way so as to achieve the same result. "Omnopon" and scopolamine in doses of one-third and one one-hundred-and-fiftieth of a grain respectively are widely used to achieve the former, and no doubt they do achieve a nicely sedated patient, but only at the expense of an unduly depressed central nervous system. Recovery is delayed and vomiting more likely. Increasing the dose of morphine from one sixth to a quarter of a grain increases analgesia and sedation by only 10%, while the same increase doubles the incidence of side-effects.

A more satisfactory method of producing an ideally sedated patient is to use a small dose of one of the antihistamines, such as promethazine (25 to 50 mg.) in combination with a smaller dose of an opiate or one of its derivatives. The antihistamines produce sedation by changing the reaction of the patient to his environment, and not by depression of the central nervous system, added to which, they enhance the action of an opiate given concurrently or simultaneously. A further advantage of the antihistamines is that they permit more liberal use of agents such as pethidine during anæsthesia, and so reduce that rather awkward immediate post-operative restlessness that so often follows the light anæsthetic techniques used today.

Choice of Anæsthetic Agent.

Obviously, in those cases in which it is considered that post-operative vomiting is likely, an attempt should be made to use anæsthetic agents which are known to be less potent causes of vomiting. Thus we may use a nitrous oxide-oxygen-relaxant technique to anæsthetize a patient, although a simpler procedure such as nitrous oxide-oxygen-ether sequence may be the anæsthetic of choice.

Post-Operative Medication.

Much the same principles apply to post-operative medication as to premedication, only more so, since the drugs used—namely, the opiates—are commonly used in higher dosages and for longer periods. When it is known that a patient is sensitive to, say, morphine, some other drug should be used, such as "Omnopon" or pethidine. When vomiting still persists regardless of the drug used, the antihistamines may be employed. Chlorpromazine or promethazine in doses of 50 mg. twice a day may be given. Promethazine is preferable, as its anti-emetic action is almost as good, while its other effects are not so widespread, as those of chlorpromazine.

Lastly, large and frequent draughts of fluids and large meals should be avoided. Even though the patient may feel like eating a large breakfast the next morning, this may well provoke a bout of vomiting.

POST-OPERATIVE HYPOPNŒA.

Post-operative hypopnœa may be defined as failure by the patient to resume adequate spontaneous respiration after a period of controlled respiration. Post-operative hypopnœa may occur from a variety of causes, but these may be conveniently divided into two groups: (i) central causes; (ii) peripheral causes.

Central Causes.

The respiratory centre is particularly sensitive to changes in the carbon dioxide level of circulating blood reaching it both directly and also indirectly via the chemoreceptor mechanism of the carotid and aortic bodies.

The respiratory centre possesses a critical threshold level for carbon dioxide. When the carbon dioxide concentration of blood perfusing the centre reaches this level, the centre fires off sufficient nervous impulses by way of the vagus to initiate a respiratory cycle of inspiration and expiration, the purpose of this being to flood the lungs with fresh air and so reduce the concentration of carbon dioxide in the lungs and in turn the concentration of carbon dioxide in the circulating blood.

This sensitive mechanism can be readily disrupted in two ways, either (i) by depressing the sensitivity of the respiratory centre so that it does not respond to normal carbon dioxide levels in the blood, or (ii) by lowering the tension of carbon dioxide in the blood so that it does not reach the critical threshold level.

The respiratory centre is depressed by most drugs used in premedication for sedative purposes—namely, the barbiturates, pethidine and morphine. It is also depressed by all anæsthetic agents, either intravenous or inhalational, with the exception of nitrous oxide. This is to be expected, as all these agents are central nervous system depressants.

The respiratory centre may further be depressed by excessively high levels of carbon dioxide in the presence of certain anæsthetic agents. This may occur as a result of hypoventilation. There seems to be some doubt as to whether an excessively high carbon dioxide concentration, such as 10% or more, in inspired air will in itself cause depression of the respiratory centre. Such concentrations will certainly cause loss of consciousness, but that is another matter.

When we go to the other extreme—namely, hyperventilation—it can easily be seen how a prolonged period of hypopnœa may follow over-vigorous ventilation. The carbon dioxide tension in the blood is reduced to a level considerably below that necessary to stimulate activity of the respiratory centre. Under these circumstances the patient's colour will remain satisfactory, because of the more than adequate oxygenation of the blood-stream, but there will be no attempt by the patient to breathe spontaneously.

Human beings exhibit extraordinarily wide variations in their individual response to drugs. It is this wide variation which may be responsible for a period of prolonged hypopnœa or even apnœa. There is no accurate means by which we may predetermine the dose of, say, thiopentone that any one patient will require. We can only arrive at an approximation from our own and from others' previous experience, and from a knowledge of the pharmacology of thiopentone under varied conditions. What we may think is an average dose for a particular patient may, in actual fact, turn out to be a gross overdosage, and may produce respiratory arrest for 15 to 20 minutes.

Finally, as a central cause of hypopnœa is what W. D. M. Paton calls "moribundity". The extremely ill or elderly patient may have reached a state where, although he is still breathing, any further insult such as general anæsthesia will bring about cessation of respiration.

Peripheral Causes.

The peripheral causes are perhaps a little more concrete, as there is only one underlying causative factor—namely, interference with neuro-muscular transmission—although there is a number of ways in which this may be brought about.

Overdosage of Relaxants.

Absolute.—I previously mentioned the wide variation in individual drug response met with in practice. Here is a further example. We normally estimate the dose of relaxant required by a consideration of several factors, such as age, sex, weight, bodily habitus, muscular development and so on. But here again, as with thiopentone, we continue to strike the occasional patient who is abnormally sensitive and reacts in a manner far beyond our expectations. The action of the relaxants is cumulative, so that once a patient is curarized any additional dose will have a far greater effect than the original dose. This becomes significant at the end of a long operation, when demands for more relaxation become pressing.

Relative.—Variations in blood flow may be responsible. At the beginning of an operation a patient may be in good condition, so that any relaxants administered are rapidly dispersed throughout the body fluids and their elimination is commenced. If the circulating blood volume is now reduced by hæmorrhage or by inadequate fluid replacement, blood flow to the muscles will be reduced, and owing to compensatory vasoconstriction, less blood will flow through the kidneys and liver. This will result in a static or even rising concentration of relaxant in the muscles, while elimination and metabolism are slowed. Thus the action of the relaxant will be prolonged.

Summation.

Ether has a similar action to curare on the neuromuscular junction, so that if the two drugs are used in conjunction, their combined effect will be much greater than if either is used alone. This may cause hypopnœa.

Abnormal Response to Relaxants.

Myasthenics.—Myasthenia is an imperfectly understood disease affecting the neuro-muscular junction, which produces a condition clinically indistinguishable from curarization. Known myasthenics should present no problem, but the so-called subclinical myasthenics will. If curariform drugs are given to these subjects, prolonged hypopnœa will result. Neonates may be classed in this group, as they are relatively much more sensitive to the competitive type relaxants than to depolarizing relaxants.

Effect of Plasma Cholinesterase.—When succinylcholine is injected into the blood-stream, it is normally and rapidly hydrolysed by cholinesterase into first succinylmonocholine and finally choline through intermediate stages. In some conditions—for example, liver disease, anæmia, malnutrition and general debility—the plasma level of this enzyme is commonly lower than normal, in which case the action of this relaxant will be prolonged. However, this may also occur in patients with a normal plasma cholinesterase level, which would seem to indicate some abnormality of the enzyme itself.

Effect of Breakdown Products of Relaxants.—Succinylmonocholine and choline have in themselves the power to cause neuro-muscular block to the extent of about one tenth to one fortieth of the parent compound. After a dose of succinylcholine of the order of 50 to 100 mg., the amount of succinylmonocholine and choline formed would not be of significance. However, if the dosage of succinylcholine is raised to 1 gramme, then these breakdown products may well cause prolonged hypopnœa.

Alteration in Type of Block.

If the depolarising relaxants are given in a high enough dosage for a long enough period, they may, apart from exerting an action through their breakdown products, change their mode of action. So, a neuro-muscular block which commeced by depolarization may continue by competition.

Irreversible Curarization.

Irreversible curarization is a condition which figured prominently in the anæsthetic Press some eighteen months or so ago. A fairly large number of cases were quoted in which relaxation had been obtained by curariform drugs, but in which, at the end of the operation, the patients had failed to respond to neostigmine. Nearly all these patients were aged over 50 years, and most were suffering from a severe illness (for example, intestinal obstruction) of some days' duration, with associated fluid and electrolyte disturbances, and they were all given large doses of curare (20 to 30 mg.). A large number of rather fanciful theories were postulated as possible causes for this. A point that seemed to me to be overlooked was that none of these patients should have been given relaxants. No one factor will suffice to explain their failure to resume spontaneous respiration. The metabolism and biochemistry of these patients are so deranged that they are in no condition to deal with a combined intravenous assault. By the use of intravenous techniques, it is not possible to grade one's doses finely enough for the requirements of patients such as these.

Treatment.

Prevention.

The possible occurrence of prolonged hypopnœa can be lessened by attention to the following factors.

Choice of Anæsthetic.—A significant fact in the occurrence of prolonged hypopnœa is that the majority of affected patients are ill or seriously ill patients. Generally speaking, a safe rule to follow is: the more ill the patient, the more simple the anæsthetic. Unfortunately the modern tendency seems to be the reverse. When there is any doubt in the anæsthetist's mind as to the safety of using relaxants or other intravenous agents for a particular case, their use should be abandoned. While the patient continues to breathe spontaneously, we can judge very finely the amount of anæsthesia required; but once that sign is abolished, the dosage required is largely arrived at by guesswork.

Where there is an obvious contraindication to the use of relaxants, such as a history of muscular weakness, our task is easy. It is when we are faced with the seriously ill, or when for some reason our suspicions are aroused, that the need for care in selection of the anæsthetic agent becomes paramount.

Test Dose.—The use of a test dose of relaxant with a delay of three to five minutes to observe its effect has been discarded for the most part, as our knowledge of and confidence in the use of relaxants have grown. However, there are still occasions (such as the bad risk cases previously mentioned) when the test dose may be of value if relaxants are to be used.

Careful Dosage.—Most of the drugs used intravenously in anæsthesia today are prepared for convenience in concentrated form, and only too frequently, and again for convenience, are administered in that concentrated form. May I make a plea here for the use of more dilute solutions? Thiopentone for many years has been administered as a 5% solution. Since the use of the 2·5% solution has become more widespread, it has become apparent that for all those years patients have been overdosed with thiopentone. I venture to suggest that similar results would be obtained with the relaxants if they were given in more dilute solutions as a routine procedure. A further aspect of this use of dilute solutions is the increased safety margin afforded, for example, when the drug is given into the wrong place, such as an artery instead of a vein, or when one drug is given in mistake for another.

Treatment of Established Condition.

It is difficult to lay down any hard and fast rules for the established condition, as every case is different, and each seems to be surrounded by a certain aura of confusion. Nobody ever really knows why it happened, while everybody always has a few suggestions to make as to what should be done. But there are a few points which may be helpful.

First, is the hypopnœa of central or peripheral origin? If the patient is making no respiratory effort whatsoever, the best course is perhaps to do nothing apart from maintain respiration and circulation and await developments. If, on the other hand, some slight respiratory effort is being made, as may well be the case, observation of the type of respiration may give a lead. If the hypopnœa is of central origin, respiration will be either very shallow and slow, or very deep and very slow (two to three breaths per minute), with no tracheal tug and no pause between inspiration and expiration. The former (shallow and slow) type occurs in thiopentone sensitivity and as a result of over-ventilation, while the latter (very deep and very slow) type occurs from morphine overdosage or sensitivity. If the hypopnœa is peripheral in origin—that is, due to overdosage with relaxants or to some abnormal response to them—the initial respiratory movements will be shallow and jerky, and there will be a distinct pause between inspiration and expiration, and tracheal tug will be present. This is Morton's "rectangular breathing".

The majority of cases will fall into one of these two categories, in which case some positive action can be taken. "Megimide" and amiphenazole can be given for barbiturate overdosage, and nalorphine for morphine or pethidine overdosage.

If the cause of the hypopnœa has been localized as peripheral, then it but remains to decide whether the residual neuro-muscular block is of the depolarizing or competitive type. If only a competitive type relaxant has been given, then atropine and neostigmine may safely be given. However, if a depolarizing relaxant has been given, the block may be either of depolarizing or of competitive type. A small dose of gallamine may resolve this. If it is of the competitive type, respiration will once again cease, in which case atropine and neostigmine

may be given; if it is of the depolarizing type, the small dose of gallamine will have no effect on respiratory movement, in which case artificial respiration must be maintained until the patient's respiration returns to normal.

Conclusion.

When one is confronted with a patient who, at the conclusion of the operation, declines to resume adequate spontaneous respiration, it is probably safer to err by doing too little rather than too much. The majority of patients, unless there has been some gross error in choice of anæsthetic agent or in dosage, will eventually resume satisfactory respiration of their own accord. The basic ætiology of the condition is inevitably associated with some degree of pharmacological confusion, and any further drugs we may give will tend to aggravate this confusion, rather than alleviate it.

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AN ALTERNATE CIRCULATORY ROUTE IN HUMAN SKELETAL MUSCLE.

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PREVIOUSLY the writer (1958a) described arterio-venous anastomoses in the pterygoid musculature. It was not possible at that time to describe the details of the arterio-venous anastomoses observed, but further investigation has enabled him to describe in some detail these structures, and the existence of an alternative circulation.

Material.

Temporo-mandibular joints with their surrounding musculature (temporal, lateral and medial pterygoids) were removed *en bloc* from several fœtuses (approximately at term). These were embedded in paraffin, so that as nearly as possible they would be cut in sagittal sections. Serial sections were cut at intervals of 12 μ. Every third section was stained with hæmatoxylin and eosin, Verhoeff's Van Gieson stain, carmalum picro-indigo-carmine stain and Davenport silver nitrate stain respectively.

Results.

The alternative strategic circulatory routes controlling the blood supply to the masticatory muscles are located in the epimysium of those muscles. The essential features of this circulation appear to be as follows (Figure I). The afferent artery (A.A.) as it approaches the muscle gives off two branches, one (M.A.) to the perimysium and the other (C.A.) to the epimysium. It can be noted that the latter artery (C.A.) originates proximal to the artery destined for the perimysium (M.A.). Approximately at the same level, two veins (M.V. and C.V.) enter the efferent vein (E.V.). Two valves (V.V.) are located at the point of entry of these veins into the efferent vein. The vein (M.V.) is probably the terminal part of an arterio-venous bridge (A.V.B.), and the vascular architecture of the direct vascular supply to the muscle seems to be arranged on the same pattern as that described for other tissues by Zweifach (1949).

The features of this circulation are seen in serial sections. The serial numbers of the photomicrographs indicate their distance from the start of sectioning. The sections run latero-medially in the sagittal plane, and they were cut at intervals of 12 μ, every third section being stained, so that the serial number 185 on the photomicrograph indicates that it is 6660 μ medial to the start of sectioning and 36 μ medial to the photomicrograph with the serial number 184.

The artery to the perimysium (Figure IV) originates 160 μ medial to the artery to the epimysium (Figure IX), and the efferent vein from the perimysium (Figure II)[1] is 128 μ medial to the efferent vein from the epimysium (Figure VI). The valves at the entry of these veins are clearly seen (Figures II, VII). The lumen of the afferent

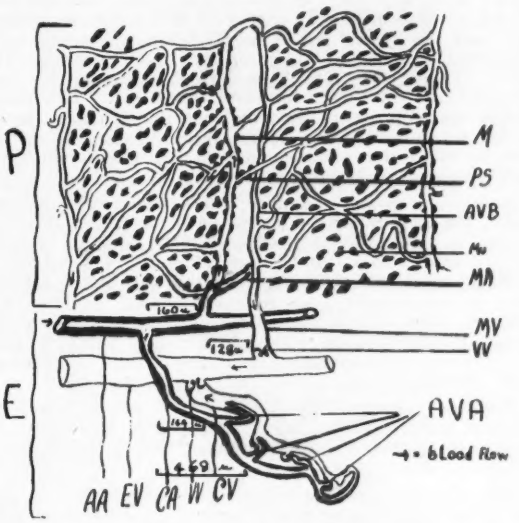


FIGURE I.

Schematic drawing of the alternative circulatory route in skeletal muscle. The afferent artery (A.A.) as it approaches the muscle gives off two branches, one (C.A.) to the epimysium (E.), the other (M.A.) to the perimysium (P.). The artery (C.A.) originates proximal to the artery (M.A.) to the perimysium. Two veins enter the main efferent vein (E.V.), one (M.V.) from the perimysium, the other (C.V.) from the epimysium. The entries of these veins to the main efferent vein (E.V.) are guarded by valves (V.V.). The circulation in the perimysium exhibits a metarteriole (M.), an arterio-venous bridge (A.V.B.) and precapillary sphincters (P.S.). Mu., muscle fibres. Arterio-venous anastomoses (A.V.A.) are a prominent feature of the alternate circulatory route.

artery decreases in size medial to the origin of its branch to the epimysium (Figures V, IX), whilst the lumen of the efferent vein increases in size. Therefore the direction of the main arterial flow is latero-medial, whilst that of the venous flow is medio-lateral.

The exact nature of the afferent blood vessel of the alternative circulatory route is difficult to assess. Near its origin, the proportionate width of its lumen to the thickness of its wall suggests its arterial nature. The histological characteristics of arterio-venous anastomoses have been described by Daniel and Prichard (1956). They possess the following morphological characters: (i) The media is unusually thick for a blood vessel of that size, and is composed of a variable number of epithelioid cells and smooth muscle cells. (ii) The epithelioid cells may be distinguished by their round, oval or polyhedral-shaped pale nuclei, cytoplasm which stains poorly with acid dyes, and cell borders which are hard to see. (iii) The smooth muscle cells of the media may consist of the typical smooth muscle cells that occur in smaller arteries, and modified

[1] For Figures II to XXII see art-paper supplement.

smooth muscle cells. These latter cells are distinguished from the typical smooth muscle cells by possessing shorter and stouter nuclei and a paler cytoplasm. (iv) The internal elastic lamina is absent.

On this basis, the afferent blood vessel of the alternative circulatory route could be classified as an artery. The endothelial cells appear to form a continuous lining of the lumen (Figure XVI), the media is not exceptionally thick and for the most part no cells project into its lumen. Near the origin of the arterio-venous anastomoses the vessel appears to exhibit some characteristics of the arterio-venous anastomoses (Figure XIII).

The first definite anastomosis with the efferent vein of this circulation occurs 144 μ from the origin of the afferent artery. There can be recognized the lumen of the artery (C.A.) and the lumen of the arterio-venous anastomosis (A.V.A.). The corrugated nature of this lumen is distinctive, and the epithelioid or modified smooth muscle cells projecting into it are exceptionally clear (Figures XIV, XV). Daniel and Pritchard (1956), speaking of this characteristic, make the following statement: "The lumen of the vessel is often far from smooth. This is shown by the irregular contours of the injection mass filling the arterio-venous anastomoses both in the Berlin blue preparations and in the neoprene casts."

Subsequent to the origin of this arterio-venous anastomosis, the afferent artery pursues a more or less tortuous course in the epimysium (Figure XVI), and an arterio-venous anastomosis can be seen (Figure XVI), which is probably a lateral section through the wall of the arterio-venous anastomosis seen in the previous section. The efferent vein of this circulation exhibits numerous red blood corpuscles inside its lumen (Figures XVII, XVIII), in contrast with the empty appearance of the main efferent vein (Figures X, XI).

Another arterio-venous anastomosis arises from the afferent artery approximately 468 μ from the origin of that artery. The cellular detail of this anastomosis is similar to that of the preceding one.

The afferent vessel of this circulation decreases in size as it proceeds through the epimysium, and eventually terminates by anastomosing with the efferent vein of the alternative circulatory route.

Discussion.

Clara (1927, 1939), Clark (1938) and Boyd (1952) have reviewed the literature on blood vessels which have been described as arterio-venous anastomoses. Daniel and Prichard (1956) have listed the sites where arterio-venous anastomoses of characteristic morphology have been found. These sites are the external ear of the rabbit, the calf and man, the human finger and toe, the bird's foot, the coccygeal body of man and the caudal glomeruli of various animals, the nasal mucosa of man and the nasal mucosa and tip of the nose of various animals, and the tongue of the dog, sheep and goat. Le Gros Clark (1958) states they exist in the mucous membrane of the gastro-intestinal tract, the thyroid gland, the carotid body and the sympathetic ganglia of man.

Brown (1937) has described the details of the innervation of the walls of arterio-venous anastomoses. She makes the following statement:

> It seems reasonable to conclude that the elaborate arborizations of thick fibres are receptor mechanisms primarily concerned with vascular reflexes in which the activity of arterio-venous anastomoses is involved. On the other hand the much thinner, probably unmyelinated fibres may be concerned with vasoconstriction since they end on the surface of the modified media of the Sucquet-Hoyer canal.

In direct observations on the living animal, arterio-venous anastomoses exhibit spontaneous rhythmic changes in calibre (Grant, 1930; Clark and Clark, 1934). They have been observed to change their calibre with temperature changes (Grant, 1930; Grant, Bland and Camp, 1932; Clark and Clark, 1934).

It appears that the function of the arterio-venous anastomoses located in exposed regions (ears, fingers, toes, etc.) is to keep them warm when exposed to cold by allowing a greatly increased blood flow through the part (Grant and Bland, 1931). Grant (1930) found that the arterio-venous anastomoses in the rabbit's ears constricted in response to adrenaline and to faradic stimulation of the sympathetic nerves, and dilated in response to acetylcholine and histamine. Tischendorf (1938) came to a similar conclusion with regard to the effects of adrenaline and histamine.

The fact that the alternative circulatory route described in this paper exists in the fœtus at term does not militate against its existence in the adult, for arterio-venous anastomoses have been found in large numbers in the ears of the sheep, not only at term, but as early as the 90th day of a gestation period of 147 days (Daniel and Prichard, 1956), in the sheep fœtus tongue at 98 days (Prichard and Daniel, 1934), and in the digits of human fœtuses of four and five months (Masson, 1937).

There seems no doubt that the circulatory route demonstrated in the epimysium of human skeletal muscle has to do with the periodic metabolic requirements of that tissue. During rest, the blood flow through the forearm muscles is about 2 to 7 ml. per 100 ml. of muscle per minute, whilst in strong rhythmic exercise the rate of flow may increase during the phases of relaxation to about 40 ml. per minute for each 100 ml. of tissue enclosed (Fulton, 1955).

The observation by Barcroft and Swan (1953) that, in subjects during a faint, although the skin was pallid and the flow of blood through the hands and feet decreased, yet the flow of blood through the muscular part of the forearm increased, suggested to them the existence of vasodilator sympathetic fibres to human skeletal muscle. However, this may not be the case, since the areas of pallor and decrease in blood flow were those in which arterio-venous anastomoses are known to exist. It follows that if the circulatory route described in this paper was under the same nervous control as the arterio-venous anastomoses in the skin and hand, then closing of this route would mean that more blood would circulate through the perimysium. Barcroft and Swan (1953), commenting on the blood flow through muscle during exercise, made the following statement:

> To sum up then release of sympathetic vasoconstrictor tone increases the blood flow through muscle, but apparently not through the same vessels as subserve its metabolic requirements. What is the anatomical basis for this?

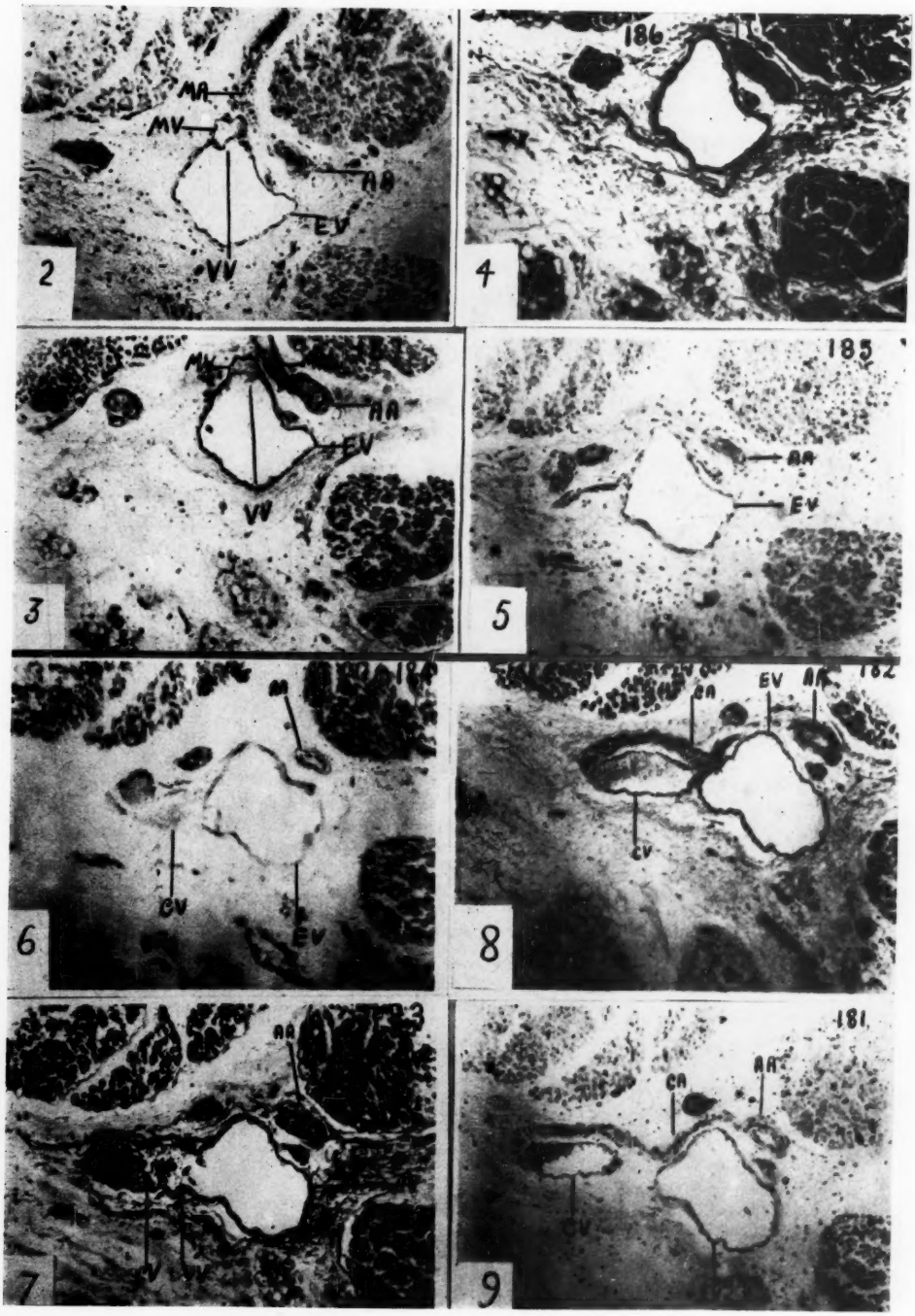
They suggested that if arterio-venous anastomoses existed in human skeletal muscle, then the features of blood flow could be explained if the vasomotor centre regulated the arterio-venous anastomoses in the general interests of the circulation and if the capillary bed was controlled by metabolites according to local requirements, and that the discrepancies between the results of plethysmographic and sodium clearance experiments could be explained on this basis. It seems to the writer that the alternate circulatory route in human skeletal muscle described in this paper is compatible with the results of physiological experiments; that is, there appear to be in skeletal muscle two vascular nets, one to subserve its metabolic requirements and one under the control of the sympathetic nervous system. The former in the perimysium and endomysium would be under the control of metabolites, the latter in the epimysium and under vasomotor control. Certain clinical evidence (Griffin, 1957, 1958*a*, 1958*b*) suggests that this latter mechanism participates in neuro-vascular reflexes.

Summary.

1. Temporo-mandibular joints with their musculature were removed *en bloc* from several fœtuses approximately at term. They were orientated as nearly as possible in the sagittal plane and cut in serial sections at intervals of 12 μ, and every third section was stained.

2. Alternative circulatory routes were observed in the epimysium of the masticatory muscles.

ILLUSTRATIONS TO THE ARTICLE BY C. J. GRIFFIN.



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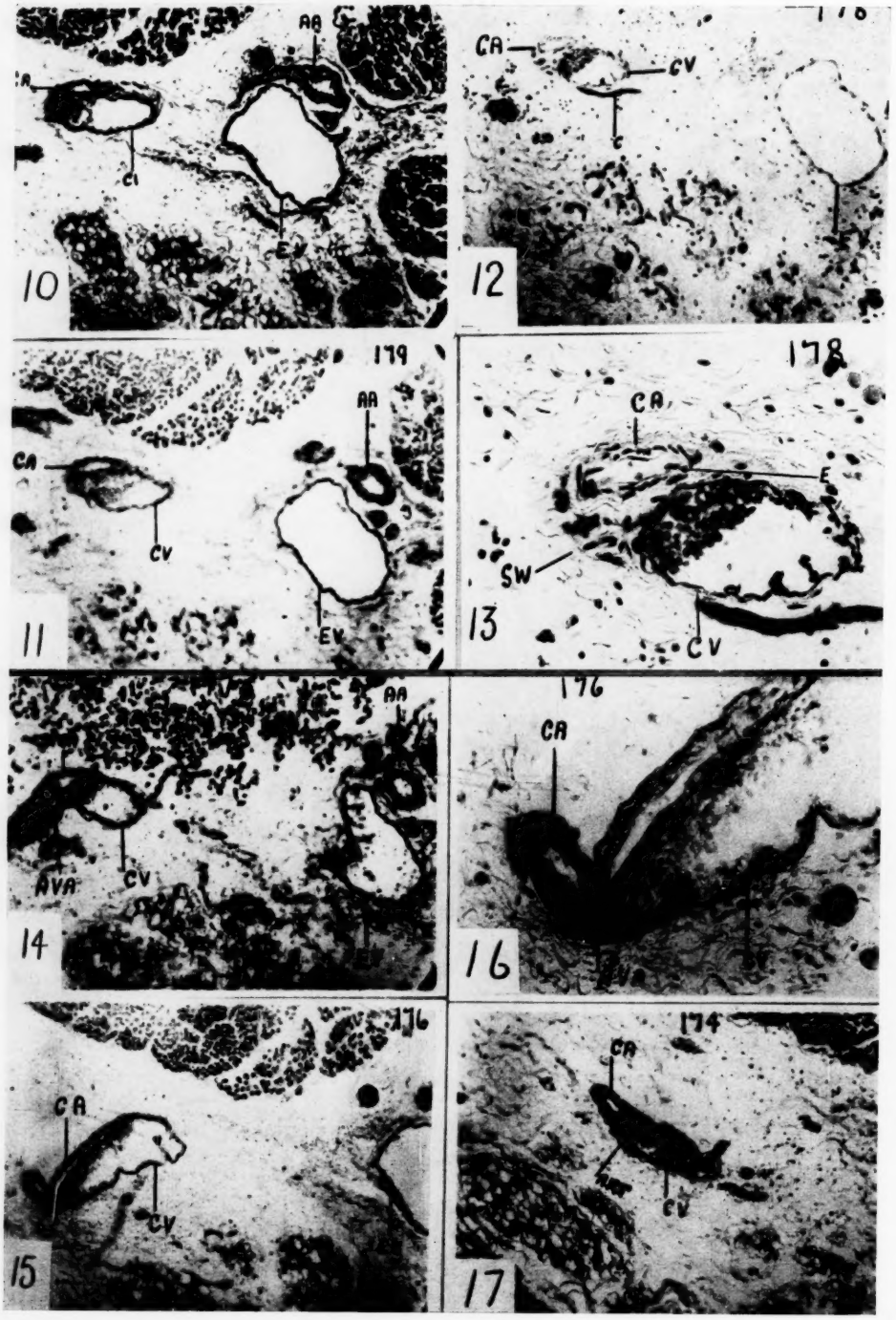

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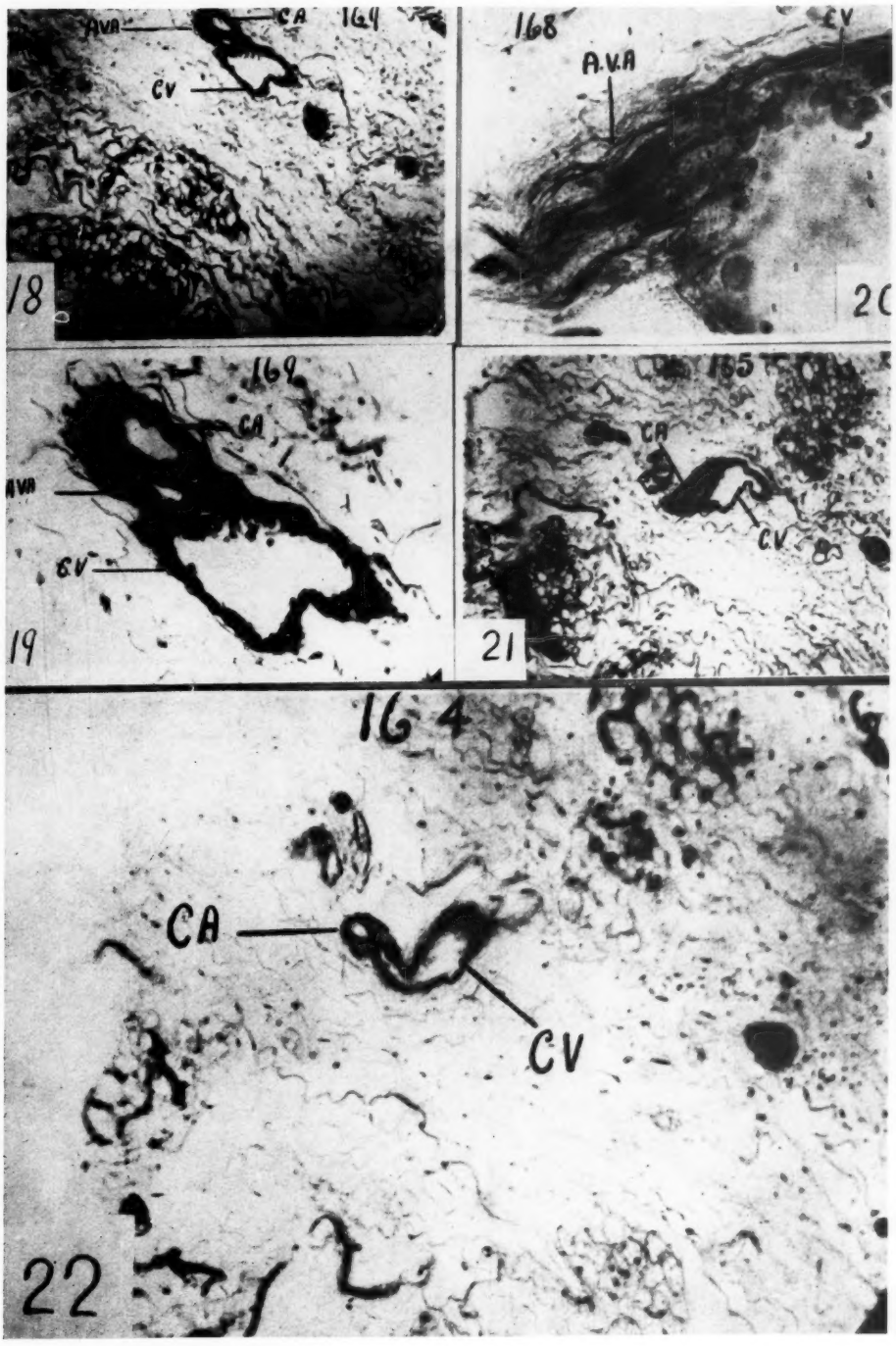

ILLUSTRATION TO THE ARTICLE BY R. T. W. REID.

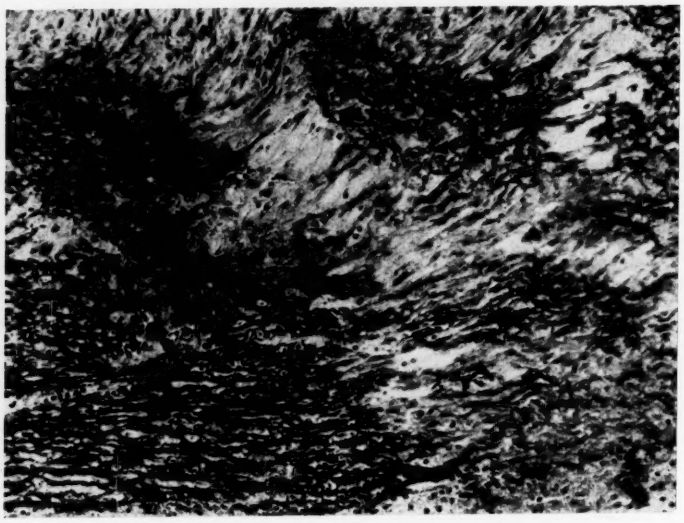

FIGURE I.
Media of aortic arch, showing irregularity and loss of elastic tissue (elastic stain, × 120).

ILLUSTRATIONS TO THE ARTICLE BY J. M. O'DONNELL.

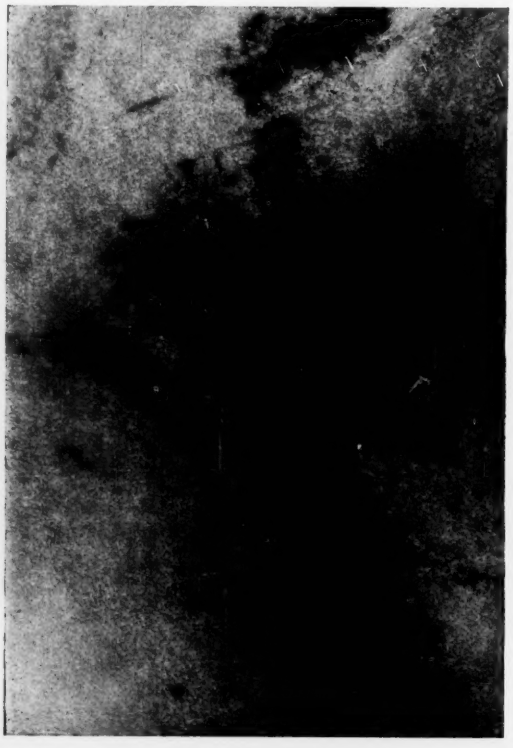

FIGURE II.

FIGURE III.

3. The location of this circulation is such as to control the blood supply to the muscle.

4. Venous valves and arterio-venous anastomoses are a prominent feature of this circulation.

5. It is suggested this circulation fulfils the requirements of physiological experimentation.

6. Certain clinical evidence suggests that this mechanism participates in neuro-vascular reflexes.

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Legends to Illustrations.

FIGURE II.—Serial section 188. The artery to the perimysium (M.A.) and the termination of the efferent vein (M.V.) from the perimysium, together with the valves (V.V.) as it anastomoses with the main efferent vein (E.V.), can be seen. (Hæmatoxylin and eosin stain, × 130.)

FIGURE III.—Serial section 187. This section is much the same as the preceding section. (Carmalum picro indigo carmine stain, × 130.)

FIGURE IV.—Serial section 186. The origin of the afferent artery (M.A.) to the perimysium can be seen. (Verhoeff's Van Gieson stain, × 130.)

FIGURE V.—Serial section 185. (Hæmatoxylin and eosin stain, × 130.)

FIGURE VI.—Serial section 184. (Davenport silver nitrate stain, × 130.)

FIGURE VII.—Serial section 183. Demonstrating the valve (V.V.) at the junction of the vein (C.V.) and the efferent vein (E.V.). (Verhoeff's Van Gieson stain, × 130.)

FIGURE VIII.—Serial section 182. The afferent vessel of the alternative circulatory route (C.A.) can be seen. (Carmalum picro indigo carmine stain, × 130.)

FIGURE IX.— Serial section 181, demonstrating the origin of the afferent vessel of the alternative circulatory route (C.A.) from the afferent artery (A.A.). (Hæmatoxylin and eosin stain, × 130.)

FIGURE X.—Serial section 180. The comparative width of the lumen of the vessel (C.A.) with the width of its wall suggests its arterial nature, although no internal elastic lamina can be seen. (Verhoeff's Van Gieson stain, × 130.)

FIGURE XI.—Serial section 179. There is very little change in the relations of the blood vessels. (Carmalum picro indigo carmine stain, × 130.)

FIGURE XII.—Serial section 178. Owing to a tear in the section, the afferent artery (A.A.) is not visible. C., capillary. (Hæmatoxylin and eosin stain, × 130.)

FIGURE XIII.—Serial section 178. The afferent and efferent blood vessels of the alternative circulatory route (C.A. and C.V.). The cells indicated by S.W. could possibly be modified muscle cells of an arterio-venous anastomosis, whereas E. may be an epithelioid cell. (Hæmatoxylin and eosin stain, × 400.)

FIGURE XIV.—Serial section 177. The definite anastomosis between the afferent and efferent blood vessels of the alternative circulatory route (C.A. and C.V.) can be seen. A.V.A., arterio-venous anastomosis. (Verhoeff's Van Gieson stain, × 130.)

FIGURE XV.—Serial section 176. The afferent blood vessel (C.A.) pursues its course through the epimysium. (Carmalum picro indigo carmine stain, × 130.)

FIGURE XVI.—Serial section 176. An arterio-venous anastomosis from the afferent vessel (C.A.) into the efferent vessel (C.V.) can be seen. It is probably portion of the anastomotic canal seen in Figure XIV. (Carmalum picro indigo carmine stain, × 400.)

FIGURE XVII.—Serial section 174. An arteriole (A.R.T.) is seen originating from the afferent blood vessel (C.A.). (Verhoeff's Van Gieson stain, × 130.)

FIGURE XVIII.—Serial section 169. An arterio-venous anastomosis can be seen. (Verhoeff's Van Gieson stain, × 130.)

FIGURE XIX.—Serial section 169. More detail of the arterio-venous anastomosis (A.V.A.). (× 400.)

FIGURE XX.—Serial section 168. Detail of the wall of an arterio-venous anastomosis. A.V.A., arterio-venous anastomosis. C.V., efferent vein from epimysium. (Carmalum picro indigo carmine stain, × 1300.)

FIGURE XXI.—Serial section 165. The afferent artery (C.A.) and efferent vein (C.V.) proceed further into the epimysium. (Verhoeff's Van Gieson stain, × 130.)

FIGURE XXII.—Serial section 164. It can be seen that the lumens of the respective vessels are reduced. (Carmalum picro indigo carmine stain, × 130.)

POLYCYTHÆMIA VERA.

By R. W. BEAL,

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A RECENT case of polycythæmia presented a number of interesting features, which prompted a review of other cases of polycythæmia vera in the records of the Royal Prince Alfred Hospital, Sydney, with particular emphasis on the clinical features and certain hæmatological findings.

Historical Survey.

In 1892 Vaquez described a case of chronic cyanosis, in which the red cell count was elevated. Saundby and Russell in 1902 wrote of a similar case; they considered that it was "a definite clinical entity, and one which was new to medical science". It remained for Sir William Osler, in 1903, to describe nine cases of chronic cyanosis, four from his own clinic, and five from the literature, under the title of "Chronic Cyanosis with Polycythæmia and Enlarged Spleen: A New Clinical Entity". He analysed these cases and made some pertinent remarks regarding the future investigation of this condition.

By 1912 Lucas was able to collect 179 cases, and in 1928 Harrop published an extensive review of the literature concerning polycythæmia up to that time.

The ætiology of this disease, at the time when Harrop wrote, as indeed it is now, was a matter for considerable

discussion. There were those who considered that it was a hyperplasia of one element of the bone marrow, analogous to leukæmia, while there were those who held that it was a response of the bone marrow to anoxæmia, there being several causes postulated for this anoxæmia. Reznikoff *et alii* in 1935 described 134 cases of polycythæmia vera encountered in a group of American hospitals, in which *48% of the patients were Eastern European Jews.* This influenced them in putting forward a theory that polycythæmia vera was in some way allied to thromboangiitis *obliterans, the incidence of which is also high in Jews* born in Eastern Europe. They were able to demonstrate in a small number of cases some histological evidence of fibrosis of the sub-intima and adventitia of the bone marrow arterioles. Others have been unable to confirm *their findings.*

Modern concepts of the pathogenesis of this disease have been greatly influenced by the work of Lawrence and his associates, and Damashek, Lawrence, Berlin and Huff (1953) published a survey of 263 patients, and explained *the high total red cell volumes by postulating two separate* populations of red cells, one with a normal life span, and one with a shorter than normal life span. They held *that this was a neoplasia-like disease, a condition* characterized by increased red-cell production, with no lengthening of red-cell life span. Damashek (1950) had *previously put forward his concept of this disease as a* panmyelopathy, which led not only to erythrocytosis but to pancytosis, and stated that an increase in the leucocytes and thrombocytes could be expected in addition to the erythrocytosis. He was one who denied the hereditary *influence in Hebrew stock, and pointed out that familial* polycythæmia vera was rare. The panmyelopathy was thought to be due either to bone-marrow stimulation by an *unknown factor or factors, or to diminution of marrow* inhibitory factors.

Wasserman (1954) summarized the pathogenetic theories of this disease, agreeing that it was a panmyelosis. However, he held that the erythrocytosis was merely an initial *facet of a complex disease process, which could ultimately* affect the bone marrow in a number of different ways.

In a recent excellent review, Pike, after weighing all the available evidence, concludes that polycythæmia vera is "a myeloproliferative disorder, probably of neoplastic *origin, in which a hyperplastic bone marrow gives rise to* high peripheral blood counts, with an increase in red blood cells as the predominant picture".

Report of a Case.

A man, now aged 54 years, was *first examined at the Royal* Prince Alfred Hospital, Sydney, on April 2, 1954, suffering from an electrocardiographically proven myocardial infarction. His blood pressure was 160/90 mm. of mercury, and the spleen was palpable approximately two inches below the left costal margin. On April 21, routine hæmatological investigation showed him to have a hæmoglobin value of 17·6 grammes per 100 ml., a white blood cell count of 8850 per cubic millimetre and a platelet count of 347,000 per cubic millimetre. *No further investigation was made,* and the patient was discharged, well, on May 8 with a final diagnosis of coronary occlusion and splenomegaly of unknown origin.

He was readmitted to hospital on August 29, 1958, com*plaining of pain in the toes of his right foot, present for* three months. The patient had also noticed that the tips of the three lateral toes on the right foot were black. On *examination of the patient, marked plethora was evident* in the face and hands, and the spleen was palpable about 3·5 inches below the left costal margin. The liver was not *palpable. The third, fourth and fifth toes of the right foot* showed evidence of gangrene. The fourth toe was the worst affected, almost the whole of the terminal phalanx being *black and extremely tender. The dorsalis pedis artery of* the right foot was palpable, and pulsation appeared to be present. The patient's blood pressure on his admission to *hospital was 150/90 mm. of mercury.* The hæmatological findings at that time were as follows: the hæmoglobin value was 21·7 grammes per 100 ml., the red blood cells numbered *7,250,000 per cubic millimetre,* the white blood cells numbered 8400 per cubic millimetre and were of normal distribution, platelets numbered 1,656,000 per cubic millimetre, the colour index was 0·75 and the hæmatocrit was 68%. Other investigations, including estimation of the blood uric acid level, revealed no abnormality.

The patient was subjected to venesection four times, 500 ml. being removed on each occasion, and each time he obtained some subjective relief. He was discharged from hospital on September 19, 1958, and returned on October 8 for injection of 7 mc. of radioactive phosphorus. At that time progress hæmatological investigations gave the following results: red blood cells, 5,510,000 per cubic millimetre; hæmoglobin value, 16·5 grammes per 100 ml.; white blood cells, *10,400 per cubic millimetre (normal distribution);* platelets, 389,000 per cubic millimetre; hæmatocrit, 53%. The gangrene had regressed, and apart from some persistent pain in the terminal phalanx of the right fourth toe, the patient was symptomatically much better than on his admission to hospital.

Two features of this case merit comment here. The first is that splenomegaly and a raised hæmoglobin level were obvious on the patient's first admission to hospital, and in retrospect it was thought that his coronary occlusion in 1954 was due chiefly to increased viscosity *of the blood and (presumably) thrombocytosis associated* with his polycythæmic state rather than to arterial disease. He has had no recurrence of anginal pain in the intervening four years, and an electrocardiogram taken during the 1958 admission showed only evidence of old infarction. Secondly, it is interesting to note that considerable subjective and objective relief of the gangrene arising from the second thrombotic episode followed venesection.

Review of a Series of 66 Cases.

Criteria of Selection.

As these cases cover a period of almost 50 years, it is impossible to adopt criteria of selection which one would adopt today. The cases included have all been diagnosed as polycythæmia vera by the honorary medical officers, and for inclusion in this series a red blood cell count of *6,000,000 per cubic millimetre or over and/or a hæmoglobin* value of at least 18 grammes per 100 ml. were required. (The criteria set by Pike are: red blood cells, greater than *6,500,000 per cubic millimetre; a hæmoglobin value, at* least 18 grammes per 100 cubic centimetres; hæmatocrit, 55% to 80%.) However, as the figures used for comparison are those obtained on the patient's admission to hospital, some patients who had obviously polycythæmic counts on *later investigation have been included, although their* initial counts were lower than the critical level, owing to the massive hæmorrhage which necessitated their *admission to hospital. No patient is included who had* signs of pulmonary or cardiac disease suggesting that the polycythæmia might have been secondary.

Incidence.

Since 1910, 66 patients with polycythæmia vera have been seen at this hospital, either as in-patients or, in the last decade, as out-patients of the radiotherapy department. *Of these, almost half have presented since 1950 (Figure V).* This increase is due in some measure to the number of asymptomatic patients referred for assessment by observant practitioners.

Age.

To take the age of the patient on admission to hospital as the age when the disease commenced is somewhat misleading, as many of these patients had had their symptoms for years before eventually being admitted to hospital. Damashek estimates that a period of five years elapses *between the onset of the disease and the first visit to the* doctor. In this group the age range is wide, there being three patients in the under 20 years group, while the *highest incidence is in the sixth decade (Figure I). A not* inconsiderable number presented in the fifth and seventh decades. The average age when the patient was first seen *at hospital was 52 years, a figure which agrees with most* series. In the series of Lawrence *et alii* (1953), the average age was 52 years, and in Wasserman's series it was 48 years.

It is interesting to note that all the young polycythæmics *showed a gross increase in their red cell mass. Two of* these patients were brothers, the first of whom presented

in 1940 at the age of 14 years, with a red blood cell count of 9,500,000 per cubic millimetre, and a hæmoglobin value of 25·9 grammes per 100 ml. The other presented in 1949 at the age of 19 years, with a red blood cell count of 10,250,000 per cubic millimetre, and a hæmatocrit of 88%. *Both these patients have since died as a result of complications of the disease, and autopsies have confirmed that the polycythæmia was primary.* The third juvenile patient was a girl, aged 12 years, who was examined in late 1957, with a considerable degree of splenomegaly, a red blood cell count of 8,860,000 per cubic millimetre, and a hæmoglobin value of 23·5 grammes per 100 ml. Full investigation revealed no condition to which the polycythæmia *might have been a secondary phenomenon.*

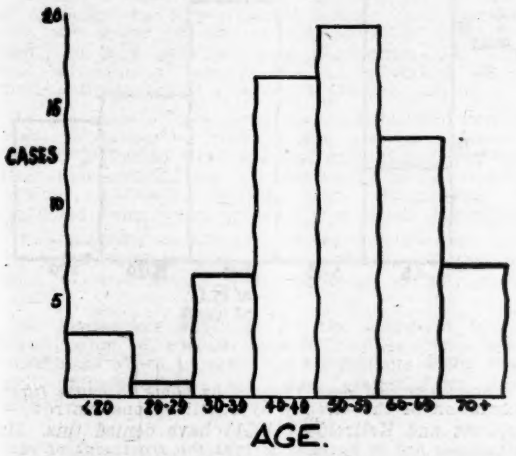

FIGURE I.

Few authors describe cases of juvenile polycythæmia vera. *Halbertsma, in 1933, described the disease in a child* of six years. Most of the large series have but one or two patients presenting under the age of 30 years. There is one patient in Wasserman's series who was aged 18 years at presentation, while among the 263 patients in the series of *Lawrence et alii*, there was one aged under 20 years. Professor M. M. Wintrobe believes that polycythæmia vera at this early age is most unusual—so much so that he *wondered whether these cases, some of which were* presented to him during his 1958 visit, may not represent a different clinical entity.

Sex.

There are 47 males and 19 females in this series, a ratio of 2·5 : 1·0. This is somewhat higher than in other series, which suggest that the ratio is nearly equal; Lawrence *et alii had a ratio of 1·32 : 1·0 and Wasserman a ratio* of 1·8 : 1·0.

Race.

Only two patients of this group are Jews of Eastern European origin.

Familial Incidence.

Apart from the two brothers mentioned above, one other *patient, an elderly female, had a sister who died from* this disease. Dr. Maurice Joseph has noted that these two brothers had bilateral pes cavus, an abnormality not seen in the other siblings, *whose blood counts were* normal.

Clinical Features.

The symptoms with which these patients presented were many and varied, but almost always could be attributed *to some feature or complication of the disease process.*

The first group of symptoms are those due to the increase *in red cell mass, and many patients have had a greatly* increased hæmatocrit. These symptoms were present in *one-third of the group. The commonest complaint of all* was headache, usually non-specific in site and character, and of considerable duration and severity. Also in this category are weakness, giddiness, tiredness and lassitude, symptoms which Osler (1904) remarked upon as being *similar to those of mountain sickness.*

A second group of symptoms were those due to enlargement of the spleen; 10% of the patients complained of either a left-sided hypochondrial mass or a dragging left-sided pain, while another 5% sought investigation of *vague abdominal discomfort.*

More than half of this hospital's polycythæmics presented with manifestations of the complications of the disease; these have been grouped as thrombosis, hæmorrhage and congestive cardiac failure. (*a*) Of all patients, *35% on one or other of their admissions, or during their* stay in hospital, showed some evidence of thrombosis; in 16% the thromboses were peripheral, mainly in the smaller vessels of the lower limbs, *14% suffered cerebral* thromboses of varying severity, while 5% had coronary artery thromboses, usually not recognized at the time as part of the polycythæmic process. (*b*) A less commonly recognized complication, but one present in 22% of all *cases, was hæmorrhage. Twelve per centum of the patients* had hæmatemeses, of whom half had proven peptic ulceration, said to be a commoner disease in polycythæmic *patients than in the average population (Wilbur and* Ochsner, 1935). The other 10% included two patients who had severe epistaxis, two with severe hæmorrhages from *tooth sockets, hæmoptysis, rectal bleeding and conjunctival* hæmorrhage. (*c*) Twelve *per centum* of the patients presented with congestive cardiac failure, usually breathless, cyanosed, and œdematous.

It is important to note that in this series, 16% of *patients were asymptomatic, and the diagnosis was made* on the results of routine hæmatological investigation.

Physical Signs.

The physical signs were few, but usually obvious.

Two-thirds of the patients showed the classical skin coloration, almost always in the face and, in many, in *the hands and the trunk as well.*

Two-thirds had splenomegaly, varying from a spleen barely palpable to gross enlargement.

One-third had hepatomegaly, a finding not often associated with polycythæmia vera.

Half of the group were hypertensive, the critical level being taken as 140/90 mm. of mercury. This finding *concurs with the figures of Lawrence et alii (1953), and* is probably equal to that of the general population of similar age selection.

Hæmatological Findings.

This review is confined to a comparison of the red-cell, hæmoglobin and white-cell values, as these are the only figures available for all patients in the group. The values *taken are those on the patients' admission to hospital.*

1. The majority had a red blood cell count (Figure II) *between 6,000,000 and 8,000,000 per cubic millimetre, and* in a significant number it was above 8,000,000 per cubic millimetre. The highest count recorded was above *13,000,000 per cubic millimetre, while the average of the* whole series was 7,500,000 per cubic millimetre.

2. The hæmoglobin value (Figure III) was most often between 17·5 and 20 grammes per 100 ml., but in half the group it was greater than 20 grammes per 100 ml., the *average being 19·5 grammes per 100 ml.*

3. Some authors describe the occurrence of leucocytosis *in as many as 75% of cases of polycythæmia vera; but in* 40% of this group the white-cell count was less than 10,000 per cubic millimetre, and in only a few was it in *the greatly elevated range (Figure IV).*

4. Hæmatocrit estimations have been carried out on *only 18 patients.* The readings range from 51% to 88%, with a mean of 68%.

5. The significance of the platelet count has been realized only in the past few years, and it has been recorded in only eight cases in this series. In two it was below 100,000 per cubic millimetre, in five between 100,000 and 180,000 per cubic millimetre, and in the other (the case reported in this paper) 1,656,000 per cubic millimetre.

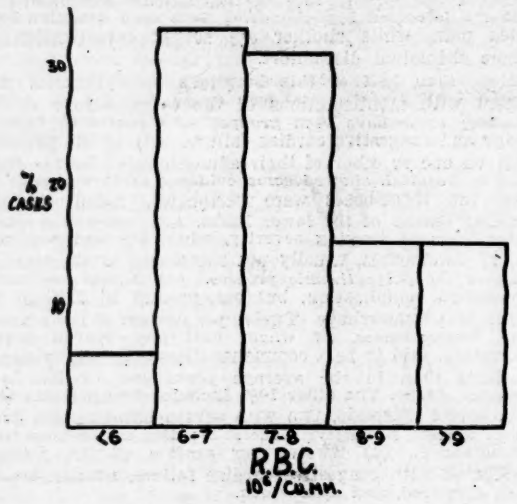

FIGURE II.

Therapy.

When Osler wrote of erythræmia, as polycythæmia vera was then termed, the treatment of the disease was venesection and irradiation of the spleen. Phenylhydrazine was introduced by Eppinger and Kloss in 1918, but has fallen from favour. The hæmolytic action of the drug certainly reduced the red-cell volume; but as the products of hæmolysis remained in the circulation, this produced a stimulus to further red-cell production by the bone marrow, thus defeating the therapeutic purpose. In 1941, Erf and Lawrence published their results of the treatment of six patients with primary polycythæmia with radiophosphorus (P^{32}). Since this time, many workers, including Stroebel, Hall and Pease (1951) and Abbatt (1954), have confirmed the excellent subjective and objective results which may now be obtained with a combination of venesection and radiophosphorus.

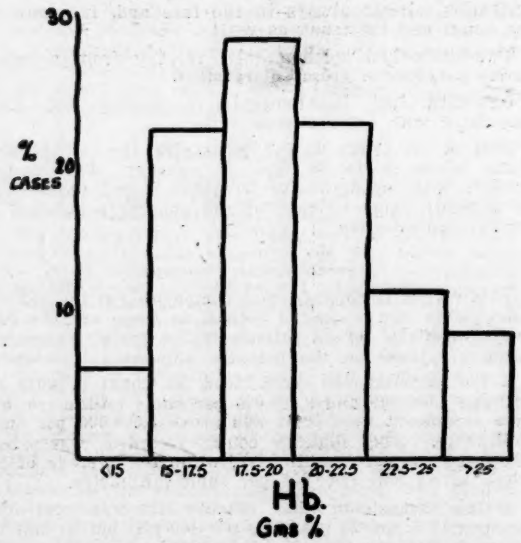

FIGURE III.

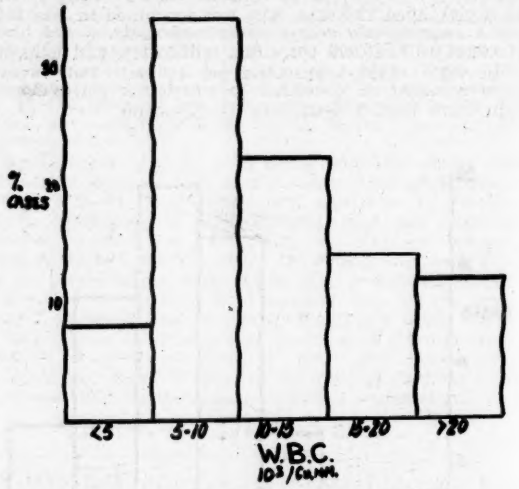

FIGURE IV.

Venesection had been thought by some to cause further stimulation of the already hyperactive bone marrow, but Stephens and Kaltreider (1937) have denied this. Most authorities are in agreement that the treatment of choice is by radioactive phosphorus with or without venesection (Lawrence *et alii*, 1955).

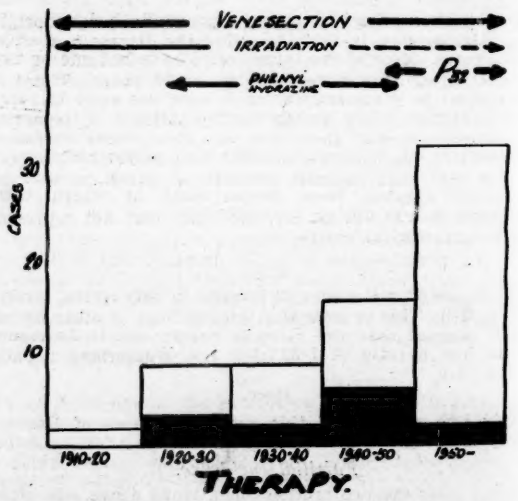

FIGURE V.

Figure V demonstrates schematically the number of cases presenting in each decade, the number in which no treatment was given being indicated in black.

All the above-mentioned methods of treatment have been in vogue at one time or another during the period of this survey in this hospital. Irradiation of the spleen and the long bones was one of the first methods introduced, and remained in favour here until about 15 years

ago. Phenylhydrazine was used from 1924 to 1948, but fell from favour with the advent of radioactive phosphorus, which, with venesection, has been the treatment of choice in the past ten years.

Ultimate Progress.

Twelve patients (18%) of this series died while in hospital, usually as a result of one of the complications of the disease. Three (5%) had massive fatal haematemeses, and five (7·5%) had thromboses which were fatal—three cerebral and two coronary. There was no evidence in any patient who came to autopsy of uterine, cerebral or renal tumours. It is not known how many of the remainder have died, or what was the cause of death of those who have died outside hospital.

Wasserman and others have pointed out the danger of operation on polycythæmic patients who, paradoxically, are prone to considerable operative hæmorrhage, owing to poor clot retraction (Lawrence, 1953). It is thus advisable to correct the hæmatological state of these patients before attempting elective surgery.

In two cases of this series the condition had developed from polycythæmia vera to the classical picture of myeloid leukæmia when the last examination was made. Two other patients not included in the series died from myeloid leukæmia, having had polycythæmia vera diagnosed some years previously at other institutions.

Two subjects at autopsy showed myelofibrosis following years of progressive splenic enlargement, the initial blood pictures having been diagnostic of polycythæmia vera.

No figures are available for the long-term follow-up investigation of treated patients, although this will be possible in years to come, as all patients being treated with radiophosphorus are being examined at regular intervals.

Summary.

1. A brief review of the history of polycythæmia vera and its pathogenesis is given.

2. The case is recorded of a patient with polycythæmia vera who suffered a coronary thrombosis in 1954 and a peripheral thrombosis in 1958.

3. A series of 66 patients with polycythæmia vera is reviewed. The average age is 52 years, the ratio of male to female is 2·5:1·0, and the mean hæmatological findings are as follows: red blood cell count, 7,500,000 per cubic millimetre; hæmoglobin value 19·5 grammes per 100 cubic millimetres; hæmatocrit, 68%.

4. Three cases presenting in adolescence are recorded, and the severity of the disease at presentation is noted.

5. A comment is made on therapy and prognosis.

Acknowledgements.

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INCIDENCE OF DIAGNOSTIC RADIOLOGY IN SOUTHERN TASMANIA IN 1956.

By J. A. BLOOMFIELD,
Hobart.

ALTHOUGH there has been great controversy about the possible hazards of diagnostic radiology, many facts remain to be discovered. A recent editorial of the *Lancet* (1958) commented: "To assess the genetically significant dose in diagnostic radiology, we need to know the number of different types of examination performed, and the age and sex distribution of those examined." In the Medical Research Council's report on "The Hazards to Man of Nuclear and Allied Radiations" (1956), it was estimated that 12,650,000 X-ray examinations were made in the United Kingdom in 1955, but this figure could only be analysed into types of examination, age and sex distribution, on figures derived from 21,000 patients attending five separate hospitals. Stanford and Vance (1955), in their analysis of the X-ray records of 9000 patients in hospitals, were unable to assess the frequency of the examinations relative to the population served by the hospitals. Similarly, Martin (1955), writing about radiation doses to the gonads, assumed "With some justification . . . that, before passing the age at which reproduction is likely, every person, male and female, undergoes a diagnostic X-ray examination other than in mass radiography". To replace this surmise with fact, the following survey was undertaken.

Tasmania, the island state of Australia, is, for many purposes, divided into two parts, the north and the south. The southern half of the island, with a population of 161,800, is a relatively closed community centred on Hobart. Much of the diagnostic radiology was performed at the three main hospitals—the Royal Hobart, Calvary and the Repatriation General. There were also in Hobart, in 1956, two radiologists in private practice. The only

TABLE I.
Analysis of X-Ray Examinations of Males.

Region or Type of Radiographic Examination.	Number of Films.[1]	Age Group (Years) and Population (December 31, 1956).						
		0 to 10 (20,800)	11 to 20 (12,500)	21 to 30 (12,600)	31 to 40 (12,300)	Total under 41 (58,200)	Over 41 (25,000)	Not Known.
Head	3	374 (1·8)[2]	318 (2·6)	321 (2·5)	243 (2·0)	1256 (2·2)	426 (1·7)	34
Cervical part of the spine	3	11 (Neg.)	16 (0·1)	43 (0·3)	47 (0·4)	117 (0·2)	220 (0·9)	1
Dorsal part of the spine	2	7 (Neg.)	31 (0·2)	35 (0·3)	38 (0·3)	111 (0·2)	109 (0·4)	1
Lumbar part of the spine and sacrum	3	24 (0·1)	77 (0·6)	173 (1·4)	195 (1·6)	469 (0·8)	557 (2·2)	5
Pelvis	1	16 (Neg.)	19 (0·1)	25 (0·2)	29 (0·2)	89 (0·2)	93 (0·4)	1
Hips	2	91 (0·4)	46 (0·2)	26 (0·2)	24 (0·2)	187 (0·3)	166 (0·7)	0
Femur, knee and leg	2	236 (1·1)	322 (2·6)	251 (2·0)	223 (1·8)	1032 (1·8)	591 (2·4)	5
Foot and ankle	2	106 (0·5)	283 (2·3)	290 (2·3)	217 (1·8)	896 (1·5)	396 (1·6)	15
Upper limb	2	621 (3·0)	834 (6·7)	587 (4·7)	414 (3·4)	2456 (4·2)	810 (3·2)	31
Chest	2	1004 (4·8)	538 (4·3)	667 (5·3)	582 (4·7)	2791 (4·5)	3952 (15·8)	123
Barium swallow	3—F[3]	18 (Neg.)	1 (Neg.)	10 (0·1)	11 (Neg.)	40 (0·1)	73 (0·3)	1
Barium meal and enema	4—F	40 (0·2)	15 (0·1)	76 (0·6)	141 (1·1)	272 (0·5)	796 (3·2)	3
Gall-bladder and bile ducts	5	—	—	21 (0·2)	38 (0·3)	59 (0·1)	165 (0·7)	1
Abdomen	2	91 (0·4)	21 (0·1)	62 (0·5)	53 (0·4)	227 (0·4)	286 (1·1)	1
Pyelography	4	15 (Neg.)	18 (0·1)	36 (0·3)	53 (0·4)	122 (0·2)	168 (0·7)	3
Miscellaneous	—	1 (Neg.)	5 (Neg.)	9 (0·1)	16 (0·1)	31 (0·1)	73 (0·3)	0
Total	—	2655 (12·8)	2544 (20·3)	2632 (20·9)	2324 (18·9)	10,155 (17·5)	8881 (35·5)	225

All figures corrected (see text).
[1] Usual number of films given for each region.
[2] Figures in parentheses are percentages.
[3] "F" indicates fluoroscopy.

other large source of diagnostic radiology was the chest survey programme of the Tuberculosis Division of the Tasmanian Department of Health. A survey has been made of the records from January 1 to December 31, 1956, of the three hospitals, of one private practice, and of the chest survey programme, and these have been analysed, where possible, into the body region examined, the type of radiographic examination performed, and the age and sex distribution.

There are possible sources of error. Several small X-ray machines were operated by doctors other than radiologists, and one private radiological practice was not included in the survey. However, allowance has been made for these (see below). There were patients who went interstate for medical investigation, or alternatively visitors to the State who required diagnostic X-ray examinations. It is known that these groups are numerically insignificant; moreover, they cancel each other. It should be noted that dental radiography has not been surveyed, except on the rare occasions when it had been performed in a department included in this study.

Collection of Data.

It is generally accepted that the possible serious hazard of diagnostic radiology is not to the individual, but to his genes. From the genetic standpoint, it is immaterial if one person is radiologically examined ten times, or ten persons once. The chances of increased mutations are the same. Therefore, examinations only have been counted; a child radiologically examined for a fractured forearm, once for diagnosis and twice subsequently for progress, has been recorded as having had three examinations.

TABLE II.
Analysis of X-Ray Examinations of Females.

Region or Type of Radiographic Examination.	Number of Films.[1]	Age Group (Years) and Population (December 31, 1956)						
		0 to 10 (19,500)	11 to 20 (11,800)	21 to 30 (11,300)	31 to 40 (11,400)	Total under 41 (54,000)	Over 41 (24,600)	Not Known.
Head	3	300 (1·5)[2]	175 (1·5)	198 (1·8)	212 (1·9)	885 (1·6)	359 (1·5)	42
Cervical part of the spine	3	9 (Neg.)	20 (0·2)	30 (0·3)	76 (0·6)	135 (0·2)	186 (0·8)	7
Dorsal part of the spine	2	4 (Neg.)	25 (0·2)	30 (0·3)	37 (0·3)	96 (0·2)	98 (0·4)	2
Lumbar part of the spine and sacrum	3	14 (Neg.)	46 (0·4)	92 (0·8)	125 (1·1)	277 (0·5)	294 (1·2)	7
Pelvis	1	7 (Neg.)	13 (0·1)	23 (0·2)	33 (0·3)	76 (0·1)	78 (0·3)	0
Hip	2	77 (0·4)	32 (0·3)	12 (0·1)	9 (0·1)	130 (0·2)	161 (0·7)	3
Femur, knee and leg	2	122 (0·6)	155 (1·3)	49 (0·4)	48 (0·4)	374 (0·7)	367 (1·5)	4
Foot and ankle	2	109 (0·6)	154 (1·3)	84 (0·7)	71 (0·6)	418 (0·8)	245 (1·0)	7
Upper limb	2	476 (2·4)	267 (2·3)	115 (1·0)	140 (1·2)	998 (1·8)	683 (2·8)	11
Chest	2	913 (4·7)	901 (7·6)	906 (8·0)	609 (5·3)	3329 (6·2)	1889 (7·7)	379
Barium swallow	3—F[3]	26 (0·1)	11 (0·1)	16 (0·1)	21 (0·2)	74 (0·1)	49 (0·2)	1
Barium meal and enema	4—F	40 (0·2)	18 (0·1)	64 (0·6)	122 (1·1)	244 (0·5)	444 (1·8)	8
Gall-bladder and bile ducts	5	1 (Neg.)	16 (0·1)	68 (0·6)	132 (1·2)	217 (0·4)	254 (1·0)	1
Abdomen	2	58 (0·3)	115 (1·0)	268 (2·4)	218 (1·9)	659 (1·2)	207 (0·8)	4
Pyelography	4	25 (0·1)	23 (0·2)	86 (0·7)	74 (0·6)	208 (0·4)	129 (0·5)	3
Salpingography	2	—	2 (Neg.)	33 (0·3)	15 (0·1)	50 (0·1)	1 (Neg.)	0
Pelvimetry	3	—	20 (0·2)	38 (0·3)	14 (0·1)	72 (0·1)	1 (Neg.)	1
Miscellaneous	—	—	2 (Neg.)	4 (Neg.)	3 (Neg.)	9 (Neg.)	18 (0·1)	0
Total	—	2181 (11·2)	1995 (16·9)	2116 (18·7)	1959 (17·2)	8251 (15·3)	5463 (22·2)	480

All figures corrected (see text).
[1] Usual number of films given for each region.
[2] Figures in parentheses are percentages.
[3] "F" indicates fluoroscopy.

The data for the survey were obtained by systematically checking through the hospital files, etc., and analysing each report card into the region examined, and the age and sex of the patient. The regions were grouped as regards the expected gonad dose (after Stanford and Vance, 1955). Thus, the skull, sinuses, mandible and mastoids were placed together as "head"; the shoulder, elbow, hand, etc., were grouped as "upper limb", and so on. The ages were analysed by decades up to 40 years; as reproduction virtually ceases after this, patients above 40 years were grouped together. Where the age had not been recorded, the examination was placed in the age-unknown column. Reference to Tables I and II shows that about 2% of the records analysed, but excluding chest survey figures, were not grouped according to age, and that the bulk of these were chest examinations. Figures supplied for the Repatriation Hospital were unsuitable for analysis as regards sex and age, and the patients were therefore assumed to be males over 40 years of age; the only exception were figures for chest examinations of staff of that hospital, which were placed in the female age-unknown column.

When the figures available had been totalled, the local firms supplying diagnostic X-ray film were approached for information regarding the total film bought in southern Tasmania and the total used in the various hospitals, etc., providing data. It was found that virtually 90% of the film supplied had been accounted for, so a correction of 10% was added. A knowledge of local conditions was used as the basis of the assumption that the examinations unaccounted for were substantially the same as those surveyed. These figures are believed to be a very accurate indication of the number of diagnostic X-ray examinations performed in southern Tasmania in 1956 (Tables I and II). Table III represents the totals of Tables I and II added to the chest survey data.

A special record was kept of all abdominal X-ray examinations of pregnant women, in addition to that in Table II, which allowed the incidence of fœtal radiation to be calculated. The following is an analysis of X-ray examinations of fœtuses in southern Tasmania in 1956.

Mothers examined	478
Examinations performed	502
Fœtuses radiologically examined	511
Fœtuses born alive	508
Total live births	3892
Percentage of all fœtuses radiologically examined	13%

The figure for fœtuses radiologically examined is higher than the number of mothers examined, because of multiple pregnancies. The figure for fœtuses born alive represents the maximum number known to be viable radiologically (records showed that one dead fœtus and two anencephalics had been detected).

Discussion.

Tables I to III show both the number of X-ray examinations performed and the percentage of the population irradiated in each age group. Perhaps the most unexpected finding is the relative constancy with which all age groups are examined. There is a general increase in the incidence of X-ray examinations with age for most regions, but the over-all pattern is different from that given by Stanford and Vance (1955), who were unable to relate their findings to the population at risk. However, there are several exceptions to this general trend, which are reflections of common experience.

Chest examinations form the largest group, not unexpectedly, as the dominant role of radiology in the diagnosis of chest diseases is well known. The abnormally high rate of chest examinations in males over 40 years of age (Table I) is probably due to the large number of patients with chest diseases being treated at one hospital. The fairly high rate of chest examinations in females aged 11 to 30 years (Table II) is also worth noting, being due to the large number of nurses undergoing routine examinations. As the yield of positive findings in nurses is so low, one examination a year would probably suffice, unless there was a special risk. The relatively high incidence of examinations of the hip in children under 10 years of age as compared with the older age groups (Tables I and II) is caused by the occurrence of congenital dislocations of the hip and by Perthes's disease. The higher incidence of gall-bladder examinations in women (Table II) reflects the more frequent occurrence of diseases of that organ in that sex. The most startling finding was that over 10% of babies born in southern Tasmania in 1956 were radiologically examined before birth, but ante-natal radiography has been drastically reduced since then.

TABLE III.
Analysis of Total X-Ray Examinations.

Sex.	Population.	Regional Radiographic Examinations.[1]	Chest Survey.		Total Population Radiologically Examined.
			Large Films.	Mass Miniature.	
Male ..	83,200	19,261	751	29,070	49,070 (59%)
Female ..	78,600	14,194	675	43,426	43,426 (55%)

[1] From Tables I and II.

No conclusion can be drawn from these figures, but they do demonstrate that people of all ages may need a diagnostic X-ray examination. It should be stressed that these figures apply to 1956, and with the recent great interest in radiation hazards, the incidence has undoubtedly changed, particularly in relation to pregnancy.

Summary.

The absence of accurate statistics for the incidence of diagnostic radiology is noted.

A comprehensive survey of the incidence of diagnostic radiology in southern Tasmania in 1956 has been made, with analysis into the body regions examined, the types of radiographic examination performed, and the age and sex distribution.

The survey shows that the incidence in the various age groups is much more uniform than previous reports suggested.

Acknowledgements.

This survey could not have been performed without the help and cooperation of many persons. For permission to analyse their records, I wish to thank Dr. K. J. Friend, the Commissioners of the Repatriation Department, the Director of Tuberculosis (Tasmanian Department of Public Health), the Mother Superior of Calvary Hospital, and the General Superintendent of the Royal Hobart Hospital. I should also like to thank the Deputy Commonwealth Statistician of the Tasmanian Branch of the Commonwealth Bureau of Census and Statistics for the population figures. I am indebted to many people for help in checking and counting the X-ray record cards, but in particular to Dr. S. Malcolm, late radiological registrar at the Royal Hobart Hospital, Sister Peters of the Company of the Little Sisters of Mary, Miss Campbell, radiographer at the Repatriation Hospital, and my wife. Lastly, I should like to thank those firms supplying diagnostic X-ray film in Tasmania for giving me the requisite data on film consumption.

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INJURY BY THE DOMESTIC LAWN MOWER.

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POWERED lawn mowers used in the home are of two main types. In one there is a series of spiral blades, which cut by a scissors action in conjunction with a fixed blade. The other type is simpler and is of more recent introduction. In it there are flat blades, set like those of a fan on the lower end of a vertical axis. The blades lie within an inch or so of the ground and as they spin they chop the grass. Users of powered mowers are inflicting injury on themselves and on others. A small survey was made to study these injuries.

Patients with injuries caused by lawn mowers, who were attending St. George Hospital during December, 1958, and January, 1959, were studied. Seventeen such injuries were seen in this two-month period. One was an injury from a mower of the older type and 16 were injuries from mowers of the second type described above.

The one injury caused by the older-type mower was a lacerated finger sustained by a woman who attempted to make an adjustment near the turning blades. The 16 injuries caused by the newer-type mower comprised an injured forehead, three cases of hand injury and 12 cases of injury to a lower limb. The forehead injury was due to an over-enthusiastic attempt to start a petrol engine. The patient struck himself on the head with the starting appliance. Of the three hand injuries, one was an electric burn caused by the handling of an exposed supply wire to an electric machine. The other two hand injuries were sustained during manipulations near the moving blades. In one of these an index finger was partially amputated and in the other there were compound fractures of two fingers. Of the lower limb injuries, two were caused by objects flung up by the blades. One woman sustained a three-inch laceration across the dorsum of her foot from a flying stone, and a man got a nail embedded in his leg. There were two other men who sustained minor leg lacerations by striking against projecting parts of stationary machines. The remaining eight patients all got their injuries by thrusting part of a foot under the machine and into the turning blades. The injury most frequently sustained in this way was a compound fracture of a toe, usually the hallux. These foot injuries were most often sustained at or just after the time of starting the motor. Several of these patients were wearing leather shoes at the time of injury, and this did not appear to give them any protection.

In this series no amputation occurred or became necessary. The longest period of disability was for two and a half months, and this was in a case of injury to a great toe, which became infected. Four other patients had to attend for dressings for longer than one month. Three patients were admitted to hospital—the one with the electric burn and two with great-toe injuries.

Healing of these injuries was rather slow. This was attributed to the rather blunt nature of the trauma, resulting in necrosis of a variable amount of tissue adjacent to the laceration. Because of this a rather generous excision of the wound was found to be an advantage during treatment.

Discussion.

The possibility of preventing these injuries is a matter of some interest. Their incidence could be reduced by better instruction of the users and by better machine design. It would seem that it is too easy to get the feet into the blades of the newer type of machine and that this type of machine does cause injury by throwing up loose objects from the ground. It is believed that improvement in machine design may reduce these dangers.

Acknowledgement.

I wish to thank Dr. D. M. Storey, Superintendent of the St. George Hospital, for facilitating this investigation.

Reports of Cases.

MARFAN'S SYNDROME COMPLICATED BY DISSECTING ANEURYSM OF THE AORTA.

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SINCE Marfan's description (1896) of a little girl with long thin extremities and muscular defects, the association of arachnodactyly and ocular and cardio-vascular abnormalities (Marfan's syndrome) has been recorded on many occasions.

Cardio-vascular disease as a cause of morbidity and death in Marfan's syndrome has become recognized more frequently (Piper and Irvine-Jones, 1926; Ellis, 1940; Rados, 1942). Aortic lesions seem to be particularly lethal. In 1943, Baer, Taussig and Oppenheimer described aneurysm of the ascending aorta in two patients with arachnodactyly and congenital dislocation of the lenses. They considered that a malformation of the medial elastic and muscular tissue preceded the dilatation. By 1951, Marvel and Genovese had found 15 examples of aortic aneurysm among 29 cases of Marfan's syndrome. Eight of these aneurysms were of the dissecting type.

Several papers on Marfan's syndrome have appeared in Australian medical journals (Ingram and Inglis, 1931; Lawes and Halliday, 1944; Lambie, Shellshear and Shellshear, 1950). None of these patients suffered from dissecting aortic aneurysm. More recently, however, Neilson and Sullivan (1956), Glass (1957) and Hurley (1959) have described examples in young people with Marfan's syndrome.

This report deals with dissecting aneurysm in a young man showing the classical features of Marfan's syndrome, including severe disease of the aorta.

Clinical Record.

An apparently healthy man, aged 20 years, experienced a sudden pain in the chest as he attempted to "smash" a tennis ball. The pain, located near the lower end of the sternum, was sharp, but did not radiate. It was neither aggravated by deep breathing nor associated with shortness of breath. He became slightly shocked, but did not lose consciousness. The intensity of the pain diminished during the evening, but it prevented sleep. Next morning the pain became severe and was accompanied by nausea and vomiting. At this stage a general practitioner noted a pulse rate of 68 per minute, a blood pressure of 115/70 mm. of mercury, and a deformity of the sternum.

The patient was admitted to the Royal Adelaide Hospital on October 19, 1958; his temperature was found to be 96·6° F., his pulse rate 50 per minute and his blood pressure 120/80 mm. of mercury. The jugular venous pressure was not raised. Although he was occasionally rolling with pain, his breathing was not distressed. The trachea was in the mid-line, and examination of the chest showed no signs of pneumothorax. The apex beat, which was described as "heaving", was in the fifth intercostal space (mid-clavicular line). A systolic murmur was heard in the aortic area, and the radial pulses were of equal intensity. The only previous serious illness was rheumatic fever when the patient was eight years old. An X-ray examination of the chest showed no significant abnormality in the mediastinum or lungs. The electrocardiogram revealed no diagnostic features of cardiac infarction.

The patient lived five hours after his admission to hospital. The pulse rate increased from 50 to 88 per minute, but without alteration in the intensity of the pain. However, sudden collapse and death occurred about 24 hours after the onset of pain.

A subsequent interview with the parents revealed additional points of interest. The mother said that the patient had worn glasses or contact lenses since he was six years old and had been much taller than boys of his

age. He had been "double jointed", which allowed him to sit in peculiar attitudes and elongate his fingers by slight traction. An ophthalmologist who had examined the patient on several occasions noted partial dislocation of the lenses of both eyes, lens opacities and a high degree of astigmatism.

No other examples of Marfan's syndrome seemed to exist in near relatives.

Necropsy.

A post-mortem examination was performed 16 hours after death.

The body measured 78 in. (198 cm.) from vertex to heel. The fingers and toes were thin and long. The skin and conjunctiva were pale, and the corneæ and pupils appeared normal on superficial external examination. The hard palate was arched and shaped like an inverted "V". The left side of the chest in the line of the costo-chondral junctions projected more anteriorly than the corresponding region on the right.

The pericardial sac contained 200 ml. of fluid blood and clot. The external surface of the heart was normal except near the aortic root, where the visceral pericardium was lacerated and the surrounding fatty tissue was infiltrated with blood.

A dissecting aneurysm encircled part of the circumference of the ascending aorta and aortic arch, but spared the origins of the arteries to head and arms. There were two aortic intimal tears. The larger was 2·5 cm. above the free edge of the aortic cusps, running parallel to them for 5·5 cm. The smaller lay just beyond the origin of the left subclavian artery, was 2 cm. long and had straight edges. It ran at right angles to the long axis of the aorta and marked the distal limit of dissection. The dissection had extended proximally to the origin of the left coronary artery, which it partially surrounded.

The aorta beyond the dissection showed a few small patches of intimal wrinkling, but no deposits of lipoid were found on macroscopic examination.

Examination of the heart showed no ventricular hypertrophy; the valve cusps were thin and the chordæ tendineæ were not thickened. There was a small foramen ovale covered by a valvular flap, and the coronary artery ostia were a little elevated (5 mm.) above the free margins of the aortic cusps. The main pulmonary artery appeared normal.

The lungs were not considered unusual, except for the absence of a right middle lobe.

Microscopic Findings.

Microscopic examination of representative sections of the aorta and common iliac arteries showed extensive structural changes involving the tunica media. These were most obvious in the ascending aorta adjacent to the dissection, but were not confined to this region. There was a general loss of the regular parallel arrangement of elastic fibres, with, in places, fragmentation or complete absence of elastic tissue (Figure I). Scattered through the media were spaces of varying size containing granular material which stained pale blue with toluidine blue. These spaces were reminiscent of the lesions described as "cystic" medionecrosis. Medial elastic defects and collections of metachromatic material were also found in the left coronary artery. The intima and adventitia of the aorta were normal.

Discussion.

Dissecting aneurysm is said to occur with greater frequency in Marfan's syndrome than in other diseases (Hirst, Johns and Kime, 1958). However, only about 25 detailed autopsy reports of dissection complicating Marfan's syndrome appear in the English and American medical literature. This figure does not give a true indication of the incidence of this complication for two reasons. Firstly, *formes frustes* of Marfan's syndrome occur, in which major skeletal and ocular abnormalities are absent and the significance of dissection is not appreciated (Goyette and Palmer, 1953). Secondly, people with Marfan's syndrome who may have died from dissecting aneurysm are not necessarily recorded in medical journals or even submitted to post-mortem examination.

Defects are common in the aorta of people with Marfan's syndrome (Tung and Liebow, 1952; Hurley, 1959). They involve usually the elastic, muscle or ground substance of the media, and are thought to lead to dilatation of the aortic valve ring, aneurysm of the aorta and dissecting aneurysm. Occasionally lesions are minimal or absent, even when aortic dissection has occurred (Hurley, 1959). Although the fundamental cause of Marfan's syndrome is unknown, it is clear that major disturbances occur in the mesenchymal and skeletal tissues. However, there are few published data on connective tissue metabolism in this disease. Recently Bacchus (1958) studied the serum mucoprotein levels in a family in which Marfan's syndrome occurred in six of eight siblings. The levels were significantly less than in control subjects. The significance of this finding has not been established, nor has its relationship to Hurley's demonstration of a normal hexosamine content in the aorta in five cases.

There is an undoubted familial tendency in arachnodactyly and Marfan's syndrome, which was noticed by Achard (1902). The majority opinion favours a dominant, non-sex-linked mode of inheritance (McKusick, 1958); but investigation of 13 affected families in Northern Ireland (Lynas, 1958) showed the complete pattern of dominant inheritance in only four pedigrees. There was, however, no suggestion of recessive inheritance and no history of consanguinity.

Restriction of strenuous exercise and exertion should be considered for patients with Marfan's syndrome, especially those with clinical evidence of aortic disease. In the case of two brothers described by Wilson (1957), as well as in the present report, death seems to have been precipitated by physical activity usually tolerated by normal individuals.

Acknowledgements.

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THE BLOCH-SULZBERGER SYNDROME (INCONTINENTIA PIGMENTI).

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INCONTINENTIA PIGMENTI is a rare pigmentary condition of the skin, which has been observed quite a number of times by dermatologists and pædiatricians in other parts of the world. The only case that appears to have been reported in Australia by a dermatologist was that of Murray-Will (1954). Because of the apparent rarity of the condition in Australia, and because there may have been other cases which have not been reported, the following typical case is placed on record.

Clinical Record.

A female child, aged 11 months (born of New Australian parents), was admitted to Princess Margaret Hospital on May 29, 1957, because of bronchitis, twitching, etc. She was born prematurely at eight months, and weighed six pounds. Two days after birth she became spastic and cyanosed and required oxygen; possible cerebral injury was suspected. There was no jaundice. The child was breast fed for six months, and sat up at nine months but did not crawl; she had made no attempt to walk. Her right arm had been weak since birth and she had no teeth. Dr. S. C. Cooke of Carnarvon, who referred the patient to hospital, reported that she had been having "seizures" several times a day; these were accompanied by facial and body twitching, and had been occurring periodically since she was six months old. The family history was not recorded. Investigation of the child's history revealed that at the age of six months she had developed crops of bullæ on the body, thighs and scalp, associated with fever. As the bullæ subsided, they left pigmented areas on the skin; from about that time she had been subject to periodical episodes of twitching and stiffening. On examination of the patient at the Princess Margaret Hospital, she presented as a well-nourished, healthy-looking baby with a slaty-chocolate rash on the trunk, the flanks, the groins and the upper parts of the inner aspects of the thighs (Figure I). The eruption was not raised; it was blotchy and "spidery" in appearance; whorls were also present. On the back it had a striated arrangement; a small bulla was present on the area on the right inguinal region (Figure II).[1] No verrucose lesions were present. Systemic examination revealed no abnormality apart from absence of teeth. A piece of skin was taken for biopsy, and the report was as follows (Figure III):

> One small area of hyperkeratosis—epidermis otherwise normal. Subepidermal capillaries not increased in number; a few, however, are dilated, others show swelling of the endothelial cells. No extravasation of red blood cells, but moderate disposition of melanin around groups of capillaries in the upper dermis. Very few histiocytes and lymphocytes present.

The condition was diagnosed as incontinentia pigmenti (Bloch-Sulzberger disease). Phenobarbital, quarter of a grain three times a day, was prescribed, and as no further treatment was indicated, the child was discharged home on June 5, 1957. Subsequently the parents reported that the rash had faded considerably.

[1] For Figures II and III see art-paper supplement.

Comment.

Incontinentia pigmenti occurs predominantly in female infants. In several series reported, the patients were practically all females. The name was given to the condition by Bloch (1926) and Sulzberger (1928), because the microscopic appearance gave the impression that the basal layers of the epidermis had become incontinent of their pigment, which, instead of finding its way to the surface to be shed in the usual way, had fallen back into the dermis, where it was lodged in chromatophores.

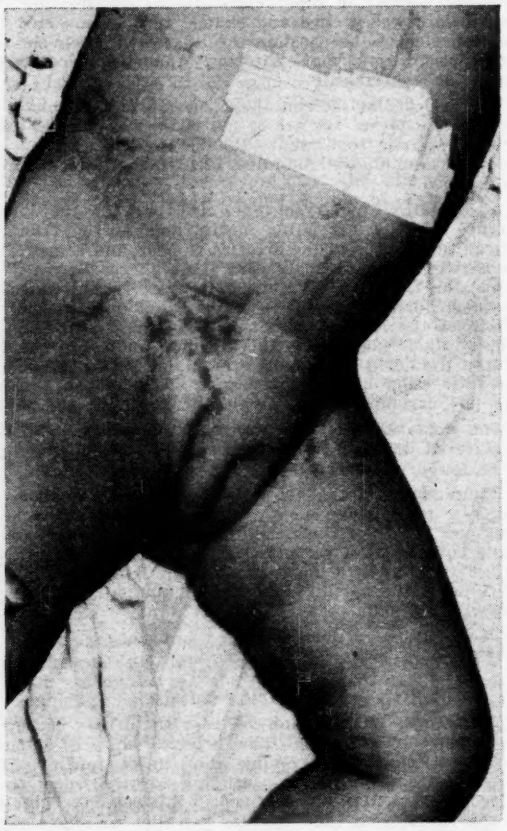

FIGURE I.

This collection of pigment in the upper layers of the dermis produces the clinical pigmentation seen in these cases. The condition may either be present at birth, or develop early in life—up to about two years usually. When present at birth, it may take the form of tense vesicles, and erythematous, papular, bullous, urticarial or lichenoid lesions. When bullous lesions are present at birth, the condition could be mistaken for epidermolysis bullosa congenita or other bullous neonatal eruptions.

The skin lesions may be associated with convulsive seizures and twitching at birth; this may create the suspicion of cerebral birth injury. The association of the nervous symptoms with any of the above-mentioned skin manifestations would help to clarify the diagnosis.

Associated with the skin picture, a large number of congenital abnormalities have been described, such as skull and other bony deformities, congenital heart disease, mental deficiency, defective dentition, absence of teeth

and so on; many different eye abnormalities, such as cataracts, corneal opacities, optic atrophy, retinal changes, etc., have been reported.

Various developmental defects in the parents and blood relations have been observed. Two cases in sisters have been reported by Rubin and Becker (1956).

Hereditary transmission has been recorded by Philpott *et alii* (1955) and by Moynahan (1954).

Because of the various anomalies associated with the skin pigmentation, Haber (1952) suggested the name "Bloch-Sulzberger syndrome" for the condition, and considered that there was no justification for retaining the name incontinentia pigmenti for it. He also pointed out that similar epidermal pigmentary incontinence occurs in the scars of poikiloderma of Civatte, lichen planus, lupus erythematosus, etc., in support of his contention. Others support Haber's view in that regard.

The usual course of skin events in the syndrome is that, when the bullæ, macules, papules, vesicles, etc., subside, the pigmentary picture presents itself with or without the preceding formation of verrucose lesions. These pigmentary changes occur at the sites of the pre-existing lesions described above and may last for years.

Philpott *et alii* (1955) reported a case in which the pigmentation was still present at the age of 27 years. The skin changes, being self-limited, eventually disappear, and are not serious as are the other associated abnormalities, such as bone deformities and optic, cardiac and other conditions.

The ætiology is obscure; many observers of the condition are of the opinion that the skin changes arise during intrauterine life. Haber (1952) suggested that it might be due to a virus infection in the mother during pregnancy. Bloom (1948) thought that the skin condition might be a "minor ectodermal defect which follows a dominant mode of inheritance". Becker (1953) regarded it a "nevoid inflammatory disturbance which appears at or shortly after birth". Epstein *et alii* (1952) believe that: "Not only is the clinical pigmentation secondary to inflammation, but the inflammation is secondary and a reaction to an epidermal abnormality which must have originated during fœtal life, if it is not genetically fixed". Kitamura *et alii* (1954) mention consanguinity, heredity and familial incidence. Naegeli regarded the skin condition as a familial chromatophore nævus.

Summary.

A further Australian case of Bloch-Sulzberger syndrome is reported, with comments and brief extracts from some of the opinions expressed concerning its ætiology. Overall it would seem that a familial and hereditary influence is operating.

Acknowledgements.

I should like to thank Dr. Neil Barsden, Honorary Pædiatrician at the Princess Margaret Hospital, for permission to report the case of this patient, who was admitted into one of his beds. My thanks are also due to Professor W. B. McDonald, who directed the investigations, to Dr. I. Lewis and Dr. P. C. Yates for their cooperation, and to Dr. S. C. E. Cooke, of Carnarvon, for his assistance in arranging the transfer of the patient to Princess Margaret Hospital for the investigations.

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RUBIN, L., and BECKER, S. W., JR. (1956), "Pigmentation in the Bloch-Sulzberger Syndrome (Incontinentia Pigmenti)", *A.M.A. Arch. Derm. Syph.*, 74: 264.

Addendum.

This patient was readmitted to Princess Margaret Hospital for Children on April 27, 1959, for further investigation. The mother said that the convulsive seizures had ceased at the age of about 16 months, and the child did not start muttering syllables till the age of about two years. She also said that, as far as she knew, there had been no similar case and no known skeletal, mental, eye or other congenital abnormalities on either side of the family; the child had been subject to crying episodes which lasted for two or three days at a time.

On examination, the child was seen to have developed fairly well, but was backward in speech, and was able to say only "mama" and a few meaningless words. She was an unhappy child, and had some peculiar mannerisms; she went off her food while in hospital. She walked badly, and there was a weakness in the right leg and arm; she used her fork and spoon with the left hand. She had crying episodes. The pigmented areas were still present on the upper and inner part of the right thigh and the right inguinal region. The skin elsewhere was free of pigmentary abnormalities. X-ray films of the skeleton revealed no bony abnormalities of the skull or elsewhere. Dr. P. C. Yates reported that there were no ophthalmic abnormalities. Chromatography of the urine revealed normal total excretion of amino acids. Six acids were resolved, cystine, histidine, glycine, tyrosine, valine and leucine, the last three in traces only. Electrophoresis revealed an increase in the gamma globulin peak, with a slight increase in the oxygen fraction. The other peaks were normal. Liver function tests gave the following results. The serum bilirubin content was 0·3 mg. per 100 ml., the serum alkaline phosphatase content 18·1 King-Armstrong units per 100 ml., the thymol turbidity 1·0 unit, and Kumkel's specific gamma globulin 2·0 units. The total serum protein content was 7·4 mg. per 100 ml. (albumin 3·5, globulin 3·9), and the albumin-globulin ratio was 1:1. The serum glutamic-pyruvic transaminase was 46 units per 100 ml. The plasma electrolyte contents, in milliequivalents per litre, were as follows: sodium 132·0, potassium 3·9, chlorides 100·5, and bicarbonate 23·0; the serum calcium content was 9·1 milliequivalents per litre and the blood urea content 32·0 milliequivalents per litre. The serum inorganic phosphorus content was 7·0 mg. per 100 ml.

Reviews.

World Health. By Fraser Brockington; 1958. Mitcham, Victoria: Penguin Books. 7" × 4¼", pp. 416, with illustrations. Price: 7s. 6d.

THIS book by Fraser Brockington, who is Professor of Social and Preventive Medicine at Manchester University, is an ambitious attempt to present the main facts and problems concerning the health of the peoples of the world in a form assimilable by the interested layman and the many individuals (including doctors) who need to know something about the global problems of health. It is written primarily from the point of view of public health and preventive medicine. The first half of the book is mainly descriptive, discussing the incidence of health and disease and the many factors, such as geography, occupation, food and so on, which influence them. The second half consists of three parts, of which the first gives an outline of public health practice throughout the world, beginning with a brief historical summary (the historical aspects of the subject are kept in view throughout this book); the second describes the development of the international approach to world health, leading up to an account of the World Health Organization, and an evaluation of the first ten years of its activity; the third discusses the use and limitations of vital and health statistics in the measurement of health. The eight appendices include the International Standard Classification of Diseases, Injuries, and Causes of Death; the International Standard Classification of Occupations; and the approved WHO programme and budget estimates for 1958. There are 15 pages of references at the end of the book, arranged to correspond

to its chapters. Professor Brockington is to be congratulated on having produced a factual outline of many aspects of world health in a compact, readable and accessible form; this volume contains much interesting detail, and should prove a very useful source of information for many purposes, with adequate references to more comprehensive sources.

Ciba Foundation Symposium on Amino Acids and Peptides with Antimetabolic Activity. Editors for the Ciba Foundation: G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.S.; 1958. London: J. and A. Churchill, Limited. 8" × 5", pp. 298, with many illustrations. Price: 45s. (English).

IN the chairman's opening remarks it is explained that when the decision to hold a symposium on amino acids and peptides was made, in order to achieve a balance between the various disciplines of chemistry, biology and clinical science, it was considered necessary to restrict the subject matter to the biologically active species. However, even with such limitations, the field was still too wide, so it was proposed to confine the attention of the symposium to amino acids and peptides with antimetabolic and cytotoxic properties. It is an indication of the rapidity of the growth of the subject that the first clear examples of amino-acid antagonists were produced after the statement by Woods and Fildes of their antimetabolic hypothesis in 1940. Seventeen discussions were held, and their scope is best illustrated by a few selected titles: "The Mode of Action of Chloramphenicol", "A Preliminary Study of the Influence of Amino Acid Deficiency on Experimental Cancer Chemotherapy", "Recent Developments in Actinomycin Chemistry", "The Synthesis of Cyclic Polypeptides", "Bacitracin", etc. As is usual in these volumes, each paper is followed by a full verbatim report of the ensuing discussion. This volume maintains the high standard of presentation and intrinsic quality which we have come to expect of the Ciba Foundation symposia.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Relationship of Blood Protein Composition to Intravascular Erythrocyte Aggregation (Sludged Blood). Clinical and Experimental Studies", by Jørn Ditzel. Acta Medica Scandinavica, Supplement 343; accompanies Vol. 164; 1959. Copenhagen: Christtreus Bogtrykkeri. 9½" × 6¾", pp. 64, with 17 illustrations. Price not stated.

From Boston University Graduate School, Department of Biology.

"Cholera", by R. Pollitzer; 1959. Geneva: World Health Organization. 9½" × 6", pp. 1019, with illustrations. Price: £5.

A comprehensive study of a disease which still constitutes a world health problem.

"A Textbook of Medicine", edited by Russell L. Cecil, M.D., Sc.D., Robert F. Loeb, M.D., Sc.D., D. Hon. Causa., LL.D.; associate editors: Alexander B. Gutman, M.D., Ph.D., Walsh McDermott, M.D., and Harold G. Wolff, M.D.; Tenth Edition; 1959. Philadelphia and London: W. B. Saunders Company. Sydney: W. Ramsay (Surgical) Limited. 10" × 6¾", pp. 1665, with 182 figures. Price: £8 5s.

The previous edition appeared in 1955.

"New Biology, No. 29", edited by M. L. Johnson, Michael Abercrombie and G. E. Fogg, 1959. 7" × 4¾", pp. 128, with illustrations. Price: 4s.

Contains seven articles on various biological subjects.

"Principles of Disability Evaluation", by Wilmer Cauthorn Smith, M.D.; 1959. Philadelphia and Montreal: J. B. Lippincott Company. Sydney: Angus & Robertson, Limited. 9" × 5¾", pp. 224. Price: 77s.

The author's aim is "to present and elaborate the philosophy and logic which necessarily must underlie any rational understanding of disability".

"La Venerologia Del Tombolo", by T. Mazzacurati; 1959. Pisa: Omnia Medica. 9¾" × 6¾", pp. 166. Price not stated.

Entirely in Italian.

"Principles of Biochemistry", edited by Abraham White, Ph.D., Philip Handler, Ph.D., Emil L. Smith, Ph.D., and DeWitt Stetten, Jr., M.D., Ph.D.; Second Edition; 1959. New York, Toronto and London: McGraw-Hill Book Company, Incorporated. 6" × 9", pp. 1164, with many illustrations. Price: $15.00.

A basic textbook and reference source for students.

"Recent Advances in Cardiology", by Terence East, M.A., D.M., F.R.C.P., and Curtis Bain, M.C., D.M., F.R.C.P.; Fifth Edition; 1959. London: J. & A. Churchill Limited. 8" × 5¼", pp. 432, with 133 illustrations. Price: 45s. (English).

Fully revised since the last edition appeared in 1948.

"Convulsive Disorders of Children", by Dora Hsi-Chih Chao, M.D., Ralph Druckman, M.D., and Peter Kellaway, A.M., Ph.D.; 1958. Philadelphia and London: W. B. Saunders Company. Sydney: W. Ramsay (Surgical) Limited. 9¾" × 6", pp. 151 with 25 figures. Price: 60s.

An attempt to provide "a concise and simple review of diagnosis, treatment and management of convulsive disorders".

"First Report on the World Health Situation, 1954-1956", Official Records of the World Health Organization, No. 94; 1959. Geneva: World Health Organization. 11" × 8½", pp. 398, with 8 illustrations. Price: 17s. 6d.

A progress report relating to WHO.

"Twelfth Report of Committee on Biological Standardization", World Health Organization Technical Report Series, No. 172; 1959. Geneva: World Health Organization. 9¼" × 6¼", pp. 44. Price: 1s. 9d.

The report of a committee which met in September, 1958.

"Second Report of Joint WHO/FAO Expert Committee on Zoonoses", World Health Organization Technical Report Series, No. 169; 1959. Geneva: World Health Organization. 9¼" × 6¼", pp. 82. Price: 3s. 6d.

The report of a committee which met in August, 1958.

"Retinal Degeneration Combined with Obesity, Diabetes Mellitus and Neurogenous Deafness. A Specific Syndrome (Not Hitherto Described) Distinct from the Laurence-Moon-Bardet-Biedl Syndrome: A Clinical, Endocrinological and Genetic Examination Based on a Large Pedigree", by C. H. Alström, B. Hallgren, L. B. Nilsson and H. Asander; Acta Psychiatrica et Neurologica Scandinavica, Supplement 129, Volume 34; 1959. Copenhagen: Einar Munksgaard. 9½" × 6½", pp. 36, with illustrations. Price not stated.

Translated from the Swedish.

"Studies on the Distribution and Fate of C^{14} and T-Labelled p-Aminosalicylic Acid (PAS) in the Body", by Åke Hanngren; Acta Radiologica, Supplement 175; 1959. Stockholm: Acta Radiologica. 9¾" × 7", pp. 118, with 53 illustrations and 10 tables. Price: Sw. Kr. 25.

The title is self-explanatory.

"Slipping Epiphysis of the Hip: A Roentgenological and Clinical Study Based on a New Roentgen Technique", by Lars Billing and Erik Severin; Acta Radiologica, Supplement 174; 1959. Stockholm: Acta Radiologica. 9¾" × 7", pp. 75, with 40 illustrations. Price: Sw. Kr. 35.

From the Department of Orthopædic Surgery, University of Gothenburg, Sweden.

"Disappearance Measurements: Theoretical, Technical, Biological and Medical Aspects", by Erik Odeblad, Björn Westin and Sven Erik Englund; Acta Radiologica, Supplement 173; 1959. Stockholm: Acta Radiologica. 9¾" × 7", pp. 70, with 21 illustrations and 7 tables. Price: Sw. Kr. 30.

The title is self-explanatory.

"Cineradiographic Studies on the Fallopian Tubes in Rabbits", by Lars Björk; Acta Radiologica, Supplement 176; 1959. Stockholm: Acta Radiologica. 9¾" × 7", pp. 54, with 13 illustrations. Price: Sw. Kr. 25.

From the Department of Diagnostic Radiology, University Hospital, Uppsala, Sweden.

"Children in Practice", by J. Peterson, 1959. London: Cambridge University Press. 8½" × 5½", pp. 234. Price: 25s. (English).

A study of the interaction between the child and his environment, with special reference to physical and mental health.

The Medical Journal of Australia

SATURDAY, DECEMBER 5, 1959.

THEORIES OF ANTIBODY PRODUCTION.

THE production of antibodies is one of the most fascinating problems in mammalian physiology, because it involves directly the modification of certain animal cells by a foreign material, the antigen, to produce novel proteins, the antibodies, which have a surface pattern complementary to the antigen used. At least, this is the basic tenet of all the so-called "instructive" theories of antibody formation. However, the problem of antibody production and antibody specificity can be looked at from another point of view. Foreshadowed by N. K. Jerne,[1] and expanded by D. W. Talmage,[2] the "selective" theory has now received comprehensive treatment in a new book by Sir Macfarlane Burnet entitled "The Clonal Selection Theory of Acquired Immunity".[3]

Burnet is no newcomer to the field of theoretical immunology. In 1941 he published a monograph[4] which for the first time looked at antibody production from a broad biological point of view, rather than from the specifically chemical viewpoint of wondering how an antigen may impose its specificity upon a globulin molecule. Eight years later he published a second edition of this monograph with F. Fenner as co-author.[5] This was noteworthy for the first explicit treatment of the problem of "self" and "not self" which is basic to antibody production. It was noteworthy, too, for its predictions on acquired tolerance, the study of which by Medawar and his group has rejuvenated immunology as a branch of biology. It may be noted in passing, but with warm approval, that Burnet's contribution to immunology and to virology has been further recognized by the recent award to him of the Copley Medal of the Royal Society.

In the present book Burnet has shifted his point of view. His own distinguished work in viral genetics made him very conscious of the concept of the clone—a population of viral particles (or cells) derived vegetatively from one parental virus particle (or cell). The introductory chapter of "The Clonal Selection Theory of Acquired Immunity" deals with clonal phenomena in bacterial and viral populations, and establishes the concepts which Burnet then transfers to his thinking about the mesenchymal cells of the mammalian body. These cells, which are among other things the scavenger cells of the body and also include the cells which produce gamma globulin, are conceived of as a vast population of cells which undergo mutation frequently, and quite by accident different mesenchymal cells produce different sorts of gamma globulin. According to Jerne's natural selection theory, antigens select appropriate natural globulins from this varied population of molecules and carry them to cells which reproduce these globulins. This idea accounts for the differentiation of self from not-self in a much more effective way than Burnet's earlier "self marker" concept, but there are serious difficulties in the view that cells receiving "selected" antibody would then proceed to produce more of this antibody. Burnet has therefore restated the "selective" theory in cellular terms. He postulates that in the animal there exist clones of mesenchymal cells, each carrying immunologically reactive sites corresponding to one potential antigenic determinant. This provides a population of cells which, at the appropriate stage of development, produce the population of globulin molecules which collectively provide the normal antibodies. When an antigen is introduced, it will contact the cell of the appropriate corresponding clone and stimulate it to proliferate, and to produce more globulin molecules of the cell's characteristic type.

Having stated the new hypothesis Burnet then proceeds to examine its implications in the essential biological phenomena of immunity: the secondary response, anamnestic responses, adjuvant action, local immunity and immunological tolerance. In the last four chapters of his book he moves beyond the confines of acquired immunity, as ordinarily conceived, into the pathology of the immune response—a field of growing importance as the severe infectious diseases become less important in our community. In these chapters various "immunological" diseases—Hashimoto's disease, acquired hæmolytic anæmias, the collagen diseases and congenital agammaglobulinæmia—are considered in terms of the clonal selection theory and its competitors, Burnet and Fenner's "indirect template" theory, and the Haurowitz-Mudd-Pauling "direct template" theory. Finally, proliferative diseases of the mesenchymal cells are discussed, and the concepts of somatic mutation and clonal selection are applied to the neoplastic diseases in general.

It is perhaps appropriate that the centenary year of the "Origin of Species" should see the publication of a book which applies the Darwinian concept of natural selection to cell populations within individual animals, rather than to populations of animals or single-celled organisms. Whatever the final validity of Burnet's clonal selection hypothesis may be, he has produced in "The Clonal Selection Theory of Acquired Immunity" a

[1] Proc. nat. Acad. Sci. (Wash.), 1955, 41: 849.
[2] "Annual Review of Medicine", 1957, 8: 239.
[3] "The Clonal Selection Theory of Acquired Immunity", The Abraham Flexner Lectures of Vanderbilt University, 1958, by Sir Macfarlane Burnet, O.M., F.R.S.; 1959. Cambridge: University Press. 8½" x 5¼", pp. 222. Price, 22s. 6d.
[4] "The Production of Antibodies", Walter & Eliza Hall Monograph 1, 1941, Macmillan, Melbourne.
[5] "Production of Antibodies", 2nd ed., 1949, Macmillan, Melbourne.

theory which invites critical experimentation (J. Lederberg[6]), and a book which in its broad sweep and persuasive argument cannot fail to stimulate all who read it.

A CANCER CONGRESS IN VICTORIA.

THE Victorian Cancer Congress, to be held in Melbourne from August 22 to 25, 1960, will in fact be much more than a State affair. However, as it is being financed by the Anti-Cancer Council of Victoria, it rightly bears the name of the State which is fathering it and which is to be congratulated on making this positive contribution to the exploration of a universal problem. A distinguished Organizing Committee has been formed with Sir William Upjohn as chairman, Professor M. R. Ewing as vice-chairman and Dr. F. Douglas Stephens as honorary organizing secretary. The Medical and Scientific Committee is under the chairmanship of Dr. T. E. Lowe. Bodies represented on these committees include the British Medical Association, the Colleges within the medical profession, the University of Melbourne, and hospitals and research institutes in Melbourne. A number of overseas authorities have been invited to contribute to the programme. It is particularly pleasing to note that Professor Rupert Willis has accepted the invitation to participate, since he is not only the accepted world authority on tumour pathology but an Australian and a graduate of the University of Melbourne. Dr. Jacob Furth, the leading authority on experimental leukæmia, has also agreed to come. The main broad topics will be carcinogenesis, leukæmia, cancer of the lung, cancer of the skin, and cancer and the community. The last-mentioned topic belongs to a session designed to bring together both the medical and the lay members of the congress to discuss important cancer problems of common interest.

Activity in the field of cancer research and control has come a long way in Australia in recent years. Particularly important in this advance have been both the Anti-Cancer Council of Victoria and the New South Wales State Cancer Council. They have sponsored and are sponsoring on quite an extensive scale research by various individuals and in various institutions in their own States, as well as programmes for the education of the medical profession and the public in respective aspects of cancer that concern them. An important activity of the Anti-Cancer Council of Victoria is the Central Cancer Registry, which maintains records of all patients suffering from cancer admitted to the major hospitals of Victoria and makes analyses of these available where they are of most value. The New South Wales State Cancer Council has taken a positive interest in the provision of facilities for treatment of cancer in medical institutions and has provided its own direct contribution to treatment as well as research in its Special Unit for Investigation and Treatment. These bodies and others throughout Australia are making a worthwhile contribution to the world-wide effort to deal effectively with the problem of cancer. The Victorian Cancer Congress will be not only a notable addition to the Australian contribution but a stimulus to further and more imaginative activity.

[6] *Science*, 1959, 129:1649.

Current Comment.

SERUM HEPATITIS.

POOLED human plasma and serum have been an enormous therapeutic asset, especially in the treatment of severe burns and injuries, and because they can be held ready for use in situations where blood is not immediately available. The only serious bugbear in their use has been the ever-present risk that serum hepatitis may result from a transfusion from a pool contaminated with the virus of homologous serum jaundice. Serum hepatitis may of course also be acquired by the transfusion of whole blood, but the risk in this case is much smaller because the blood is not pooled. According to J. G. Allen and his colleagues,[1] estimates of the incidence of serum hepatitis among patients who have received a transfusion of pooled plasma vary from 2·4% to 12%. In a more recent paper C. M. Kunin[2] analyses the incidence of serum hepatitis after whole blood transfusions given in two hospitals in the Boston area. He found an incidence of 0·11 and 0·31 per 100 pints transfused in these two hospitals, and quotes a series of other reports from various countries in which this figure varies from zero to 1·51. Kunin made the interesting discovery that in his series this incidence varied according to the source. In the United States much of the blood and plasma used in transfusions is supplied by commercial blood banks, and in the two hospitals concerned blood had been received from five different sources. Kunin's figures plainly showed that an unduly high proportion of patients who developed serum hepatitis had received blood drawn from one particular bank, and it is suggested that this bank's methods of donor screening were inadequate. He comments: "Obviously the policies of such a bank need to be reviewed; until then limited use should be made of blood from this source."

There is no way of detecting the presence of the virus of serum hepatitis in serum except by transmitting the infection, and various methods have been suggested as a means of minimizing this risk. One is to keep to pools as small as practicable, since one donor with circulating virus will contaminate the whole of the pool to which he contributes. Another is irradiation with ultra-violet light, which has been claimed by some to be effective in neutralizing the virus. However, the risk of serum hepatitis has been one of the major factors prompting the present trend towards the use of human serum albumin, which does not carry this risk, in place of pooled serum or plasma. Dr. R. J. Walsh informs us that in New South Wales the Red Cross Blood Transfusion Service encourages the use of serum albumin in preference to pooled serum and now issues about 10 bottles of the former for every one of pooled serum used. However, large quantities of pooled serum or plasma are still used elsewhere, and there may be various reasons why it is not practicable or desirable to abandon altogether the use of pooled serum or plasma in favour of serum albumin. Some years ago, considering the problem of serum hepatitis, workers at the University of Chicago Blood Bank concluded that the then current and generally employed methods of storing plasma were highly favourable to the preservation of viruses, which can often survive for long periods at low temperature, but mostly are rapidly inactivated if left at warm room temperatures. They therefore decided to store their plasma in a room whose temperature ranged from 76° to 96° F. (24·4° to 35·5° C.) and averaged 89° F.; the plasma was stored in a liquid state at these tem-

[1] *J. Amer. med. Ass.*, 1950, 144:1069 (November 25).
[2] *Amer. J. med. Sci.*, 1959, 237:293 (March).

peratures for about six months before use. This method of storage has previously been described by J. G. Allen and his colleagues in their paper already mentioned, and elsewhere. W. A. Sayman *et alii*[3] now report the experience of this bank with the operation of this routine over a period of 15 years. A six-months follow-up was carried out for all patients who received transfusions of serum only or of serum and blood, and for a 21% random sample of those who received blood only. In each category, the follow-up was 98% complete. If we exclude those who had died from other causes within six months of their transfusion and without developing jaundice, there were 305 patients in the first category, 653 in the second, and 1894 in the third. From the number who developed serum hepatitis after transfusion of whole blood only, it was possible to estimate the number who might be expected to develop serum hepatitis after receiving transfusions of plasma, if there was 100% survival of the virus in plasma from infected bottles of blood. In the event, none of those who had received plasma only developed serum hepatitis, against an expected figure of 48; also, of those who received both blood and plasma, only 21 developed serum hepatitis against an expected 123. The experience of 21 cases compares with an expected figure of 24 as a result of whole blood transfusion, if no plasma had been given. (Incidentally, the Chicago experience of serum hepatitis after whole blood transfusion works out at almost 0·6 per 100 pints of transfused blood.) These figures would appear fully to support the claim of the University of Chicago Blood Bank to have discovered a method of plasma storage which appears to eliminate, at least to a very great extent, the risk of serum hepatitis from this source.

Some additional points about the Chicago workers' report should be mentioned. The routine adopted was arrived at by chance, the conditions of storage having been in the first instance imposed by necessity; only when the welcome freedom of their patients from serum hepatitis was realized were these conditions adopted as a matter of policy. Secondly, storage at these temperatures does not apparently impair the therapeutic value of the plasma in other respects. Finally, the maintenance of a relatively high storage temperature is necessary. This was clearly brought out when on one occasion it was necessary to use plasma from another bank, which had been stored for seven months at 72° F; no less than seven out of the 42 patients who received plasma from this source developed hepatitis with jaundice.

A VARIANT FORM OF ANGINA PECTORIS.

As well as in the classical Heberden's angina pectoris, cardiac pain may be observed in myocardial infarction and in angina pectoris due to disturbing dreams, tobacco, heart failure and hypoglycæmia. Characteristics of Heberden's angina pectoris are that the pain is provoked when the work of the heart is increased and relieved by rest or the administration of nitroglycerin, and that an electrocardiogram taken during pain usually shows depression of the *S-T* segment in test leads I, II, III and V_4 without reciprocal elevation.

M. Prinzmetal, R. Kennamer, R. Merliss, T. Wada and N. Bor[1] have described another type of angina pectoris which appears to be quite common. It does not show the two main characteristics of the classical form and has other important clinical differences. In this type the pain comes on with the subject at rest or during ordinary activity during the day or night. It is not brought on by exertion. During an attack the *S-T* segments are sometimes elevated for a short time to a considerable extent, and there are reciprocal *S-T* depressions in the standard leads. The attacks usually end spontaneously but may lead to death. Prinzmetal and his colleagues have studied 32 such cases and have found reports of 12 more in the literature of cardiac pain. A typical case history showed the following features. Pain occurred with the patient at rest. Marked exertion produced no pain. The electrocardiogram was normal in the absence of pain. The results of exercise tests were within normal limits. The attacks occurred at about the same time every day. The bouts of pain occurred rhythmically. Administration of nitroglycerin terminated the recurrent pain and the electrocardiographic abnormalities. Six months after onset myocardial infarction occurred, and the infarct was at the same site as the previous attacks of ischæmia. There was immediate cessation of rest pain after infarction.

Coronary atherosclerosis is common to both forms of angina pectoris, but there is no constant relationship between the extent of the atherosclerosis and the severity of anginal pain. Myocardial infarction is common in the variant form of angina pectoris, and Prinzmetal and his colleagues conclude that atherosclerosis of a single large coronary artery is the major pathological feature of both the angina and the later infarction. It is suggested that this variant type of angina results from temporary occlusion of a large diseased artery with a narrow lumen due to a normal increase in the tonus of the vessel wall. Intermittent occlusion of a large coronary artery in a dog reproduced all the major clinical and electrocardiographic features of the attack. Nitroglycerin, which relieves arterial and arteriolar hypertonus, promptly relieves the attack. It has also been found that administration of nylidrin hydrochloride prevents attacks. This drug induces vasodilatation of sympathetic origin and is said to be contraindicated in severe Heberden's angina, for it tends to increase cardiac output and may precipitate attacks. The general treatment of the variant form is the same as that for the classical form with more use of anticoagulants in the hope of preventing infarction. The variant and classical forms of angina pectoris may occur in the same patient at the same time.

[3] *J. Amer. med. Ass.*, 1958, 168:1735 (November 29).
[1] *Amer. J. Med.*, 1959, 3:375 (September).

CYTOLOGICAL DIAGNOSIS OF MEASLES.

IT has been established that a highly characteristic feature of measles is the appearance of large multinucleated cells, particularly in the lymphoid tissues. They occur also in the sputum and can readily be recognized in mucus aspirated from the back of the nose in early cases of measles. A. J. Beale and W. Campbell[1] have described a method for examining nasal mucus for these syncytial cells as an aid to the diagnosis of measles before other signs appear. The mucus is collected from the posterior turbinate region by passing a glass pipette along the floor of the nose and aspirating the mucus. If smears are fixed immediately in ether-alcohol mixture, they can be stained satisfactorily with Papanicolaou's stain or with Giemsa stain, or even with methylene blue. In a florid case of measles in the catarrhal stage the mucus is full of the characteristic giant cell masses. Later on the giant cells break up. Material taken up to five days before the onset of the rash contains easily recognized giant cells.

Although measles is not hard to diagnose, occasions arise, particularly in children's hospitals, when it would be most valuable to be able to make a diagnosis in the catarrhal stage, and this method is useful for this purpose. There are two sorts of giant cells seen in association with measles. The epithelial giant cell or syncytium is most common in the respiratory tract, and it is these cells desquamated that enable the cytological diagnosis to be made. These cells may also occur in the bladder and on other epithelial surfaces. So far as man is concerned, these cells seem to occur only in the presence of measles. The other type of giant cells, the reticulo-endothelial type, occurs in other tissues—for example, the appendix. Giant cells occur in association with other virus diseases, but they are different, having fewer nuclei and less protoplasm.

[1] *J. clin. Path.*, 1959, 12:335 (July).

Abstracts from Medical Literature.

NEUROLOGY AND PSYCHIATRY.

Stress and the Precipitation of Acute Intermittent Porphyria.

E. D. LUBY *et alii* (*Psychosom. Med.*, January-February, 1959) describe six patients who became ill at a time when either anxiety or depression was produced by disturbing life situations. In three of these the acute episode of porphyria followed immediately after the emotional stress, and in a most dramatic manner. The mean porphobilinogen level was determined in one family group over a period of 12 weeks, and there was a close correspondence with its fluctuation and the presence of emotional stress. At a time when one member was moribund in a respirator the porphobilinogen excretion of the family rose dramatically, and as he improved it gradually diminished. The personality configurations of these patients and their families, as assessed by the Minnesota Multiphasic Personality Inventory, showed no common pattern. The authors found that pethidine, together with ACTH and chelating agents, had been of greatest value in treatment, but cautioned about the danger of addiction to pethidine.

Pictures during Mental Disturbance.

R. W. PICKFORD (*Scot. med. J.*, December, 1958) has selected 12 paintings, produced by a schizophrenic patient who had had no training or guidance in art, and who did not produce them under any psycho-analytical or other influence. Various kinds of phantasy material emerge, illustrating early oral relationships with the mother, late oral relationships and conflicts, problems about the parents, family conflicts, and finally the re-establishment of mental harmony and balance. The paintings display considerable artistic talent, and the author comments that the patient has achieved in this way what a psychotherapist is always trying to do, namely to have the patient realize his unconscious phantasies symbolically and bring them into the scope and control of his ego. He states that the essential difference between these paintings and the great works of creative art lies in the manner in which the phantasy material is handled; in the great artist these are integrated, controlled and harmonized in ways impossible for the disturbed or psychotic personality. The author concludes that if we attribute psychotic breakdowns to weakness of the ego, then creative artistic work, however crude, must always be valuable for mental patients, by manipulating and organizing the phantasy material and superego tendencies, and by helping to reestablish balance and control.

The Communication of Suicidal Intent.

E. ROBINS *et alii* (*Amer. J. Psychiat.*, February, 1959) interviewed the family, relatives, friends, workmates, doctors and ministers of 134 consecutive suicides a short time after the event. Over two-thirds of these patients had communicated suicidal ideas, and 41% had specifically stated that they intended to commit suicide. In the majority the suicidal communications were of recent onset, repeatedly stated and expressed to many people. The authors considered that 98% of these patients were clinically ill prior to their suicides. The frequency of expression of suicidal ideas was not significantly related to age, sex, marital state, religion, clinical diagnosis, occupation, income, education or whether living alone or not. Chronic alcoholics had a somewhat greater tendency than the others to make the specific statement that they intended to commit suicide.

A Mental Syndrome Associated with Lung Carcinoma.

G. P. MCGOVERN, D. H. MILLER AND E. E. ROBERTSON (*A.M.A. Arch. Neurol. Psychiat.*, March, 1959) describe two cases in which mental symptoms occurred in association with bronchial carcinoma uncomplicated by cerebral metastases, and in both cases the mental symptoms preceded the diagnosis of the growth. The psychiatric symptoms included depression, intellectual impairment and alterations in consciousness. As each of these components tended to be variable in degree, and fluctuated in their course, this suggested to the authors that there might have been an underlying biochemical disorder. However, the only positive laboratory finding was a raised blood ammonia level in one of the patients. Both cases came to autopsy, and no structural lesion of the brains was demonstrated. The precise relationship which exists between carcinoma and neuropathies is thus still unknown.

The Cholesterol and Cholesterol Ester Content of Cerebro-Spinal Fluid in Neurological Diseases.

J. B. GREEN *et alii* (*J. Neurol. Neurosurg. Psychiat.*, May, 1959) discuss the cholesterol and cholesterol ester content of cerebro-spinal fluid in patients with multiple sclerosis and other neurological diseases. The cholesterol and cholesterol ester content of the cerebro-spinal fluid was determined in six normal subjects, in 13 patients with multiple sclerosis, and in 34 patients with other neurological diseases. While the levels of free and esterified cholesterol were raised in a variety of neurological diseases, only multiple sclerosis was characterized by a proportion of ester which was over 60% of the total cholesterol content of the cerebro-spinal fluid. This finding correlates well with previous investigations of demyelinated tissue by Cumings. It is concluded that the determination of the percentage of esterified cholesterol in the cerebro-spinal fluid is a promising adjunct in the diagnosis of multiple sclerosis.

Skull Trauma and Cerebral Tumour.

J.-A. CHAVANY (*Presse méd.*, April 25, 1959) reviews the association of injuries to the skull and cerebral tumours, and presents six cases. He concludes that, although the two conditions are often found together, it is only very rarely that there is any causal connexion between them. The conditions which may permit skull trauma to affect the development of a neoplasm are stated to be: (i) hæmorrhage in the tumour at the time of the injury; (ii) weakening of the patient's general condition by the accident; (iii) hydroelectric alterations in the brain brought about by the trauma, producing round the tumour a favourable area of œdema and collapse, and through the disordered metabolism leading to the development of neoplastic tissue. The author considers that the first condition should be eliminated by operation and the second by clinical examination. The third must be taken seriously and requires further investigation. It frequently happens that a history of trauma obscures for a time the true diagnosis of tumour. The problem has medico-legal aspects.

Neurological Aspects of Leptospirosis.

R. W. ROSS RUSSELL (*J. Neurol. Neurosurg. Psychiat.*, May, 1959) discusses the neurological aspects of leptospirosis and reviews 64 cases. He states that there are four main groups of neurological disorders occurring in the disease, as follows: (i) Meningitis, which may appear early or late in the disease. (ii) Peripheral nerve lesions, usually a late complication, affecting the proximal muscles of the upper limbs. There is a close resemblance to anterior poliomyelitis or neuralgic amyotrophy. (iii) Cortical and cranial nerve lesions. These are rare and usually transient, affecting ocular, facial or palatal nerves. (iv) Spinal cord lesions. These are also rare, and consist of transverse or ascending forms of myelitis.

Clinical Appraisal of Drug Action.

E. C. KAST AND J. LOESCH (*Psychosom. Med.*, May-June, 1959) comment on the limitations of placebo control and double-blind techniques in the assessment of drug action. They studied 20 patients intensively over a period of 17 weeks, observing particularly their interpretation of the physician's management and their reactions to it. All patients suffered from anxiety neurosis with gastro-intestinal symptoms, and were treated with a combination of meprobromate and an antispasmodic. For the first six weeks they were given the drug together with enthusiasm and superficial psychotherapy; during the next six weeks the doctors' attitude did not change, but a placebo replaced the active substances. In the final five weeks the drug was administered again, but this time the doctors expressed doubt as to its action and no longer handled the patients with solicitous care. Eighteen of the patients improved from a digestive as well as an emotional viewpoint in the first phase; six could not be evaluated in the second and third phases because of changes in the life situation; three and five (emotionally and in relation to digestive symptoms) maintained improvement in the second stage and 13 and 14 in the third stage. From this the authors concluded that the drug had a significant truly pharmacological action. The results also substantiated the deep influence of the doctor-patient relationship on the efficacy of even a potent drug

PHYSICAL MEDICINE AND REHABILITATION.

The Patient after Hemipelvectomy.

T. F. CHILDS AND M. HOLTZMAN (*Arch. phys. Med.*, June, 1959) discuss the post-operative management and rehabilitation of the patient who has undergone hemipelvectomy. They state that in these cases the greatest problem is the initial and maintained fit of the prosthesis. The usual result is for the patient to walk stiff-legged; however, it is possible to develop good gait patterns with flexion of the knee of the prosthesis. The authors give a warning that these patients should not be permitted to become too adept in the use of crutches; this will cause a poor gait pattern, keeping the centre of gravity over the remaining extremity even after the prosthesis has begun to be used. They describe in detail the routine of bandaging and exercise which they recommend in the care of these patients.

Resident Training in Physical Medicine and Rehabilitation.

E. W. JOHNSON, R. E. WORDEN AND R. D. BURK (*Arch. phys. Med.*, June, 1959) state that the continued growth of physical medicine and rehabilitation depends on an increasing supply of well-trained physiatrists, which in turn depends on standardized, well-rounded resident training programmes. The field of the specialty is constantly changing, and resident training programmes should be adjusted to the changing needs. The authors suggest that seven areas of emphasis should be common to all such programmes. They are: (i) basic research facilities; (ii) comprehensive clinical training; (iii) teaching responsibilities; (iv) a bed service assigned to physical medicine and rehabilitation; (v) a broad rehabilitation experience; (vi) basic science instruction; (vii) a continuous three-year programme, in which the preceding six areas are present. The authors believe that the seven requisites are met most adequately in a university setting. They emphasize that the physiatrist must be a well-trained physician as well as a specialist, and that the diagnostic aspects of physical medicine should be stressed.

Athetosis.

H. KABAT AND M. MCLEOD (*Arch. phys. Med.*, July, 1959) state that paresis of voluntary movement is a constant finding in athetosis. Isotonic contraction is weaker than isometric contraction, and muscle power is less in the lengthened position. Imbalance of antagonistic muscles is also present. Athetoid irradiation results from irradiation of nerve impulses in an abnormal manner from attempts at voluntary motion. The greater the paresis of the prime mover, the more intense is the athetoid irradiation. In many cases spontaneous athetosis is absent at rest, but athetoid irradiation is found as a routine. When spontaneous athetosis is present, it is exaggerated by athetoid irradiation. Athetoid irradiation is abnormal in character, resulting in repetitive, purposeless movements of vermiform configuration. The irradiation patterns are abnormal, and impulses may be directed into muscle groups not normally associated with the prime mover. The irradiation is abnormally timed, producing asynchronous patterns of motion. Adequate testing of neuromuscular function requires assessment of isotonic, isometric and eccentric function in every part of the range of motion. In addition, observation of athetoid irradiation and imbalance of antagonistic muscles, and of response in irradiation patterns, is an essential component. The goal of treatment is correction of paresis with improvement in voluntary motion and performance, which is accompanied by decrease in athetoid irradiation. Modified techniques of proprioceptive facilitation are applied for this purpose, with emphasis on isotonic function and imbalance of antagonists. The athetoid irradiation resulting from the resistive exercise can safely be ignored. Training in normal patterns of movement is included in the facilitation programme. Habit training in posture, gait and essential skills is also carried out. The authors state that the results of this programme of treatment in improving voluntary motion and performance and in reducing athetosis have been gratifying.

Medical Contributions in Vocational Evaluation.

W. C. GORTHY *et alii* (*Arch. phys. Med.*, June, 1959) discuss the importance of medical participation in a vocational evaluation programme, on the basis of a study of 534 patients at the Institute for the Crippled and Disabled, New York. This work showed that the specific vocational objectives were influenced by medical recommendations for 47% of the patients who received vocational evaluation. Medical services and personnel have their greatest emphasis in specific job areas. This aspect requires physical assessment in terms of the demands of the job, the determination of limits of work tolerance, the prescription of treatment that will improve capacity for a specific job, and the development of adaptive devices that will improve job performance. The authors describe an organized system of vocational evaluation in use at the institute. They state that this system, which includes an extended period of "tryout" on work samples, provides a laboratory in which medical personnel, as well as social service and psychological personnel, can observe the performance of their patients. "Reality" testing under such circumstances further substantiates the diagnostic and evaluative observations previously made in a clinical setting.

Radiographic Evaluation of Hip Flexion Contractures.

L. M. ROTHMAN *et alii* (*Arch. phys. Med.*, August, 1959) state that flexion contracture at the hip joint is found as a complication in a wide variety of physical conditions encountered in a rehabilitation service. This complication is a frequent factor limiting the ambulation goals of the patient. The usual methods of estimating the presence and degree of hip flexion contracture by the eye alone, or even with a goniometer, are subject to considerable error. The authors state that a technique of mensuration utilizing a lateral X-ray projection has yielded reproducible measurements with a high degree of accuracy. Fixed bony landmarks give origin to lines which, when extended, form an angle. The measurement of this angle gives an absolute figure which represents the relationship between the bony pelvis and the femur, thus permitting objective evaluation of the effectiveness of therapy in dealing with limitation of hip extension. It is not suggested that radiographic mensuration should replace goniometric studies; but this method is designed to provide the investigator with a reliable method of mensuration.

Brief Isometric Exercises.

W. T. LIBERSON AND M. M. ASA (*Arch. phys. Med.*, August, 1959) have investigated, in 26 normal subjects, the effectiveness of daily brief (six-second) exercises as compared with the DeLorme exercises. The hypothenar eminence was exercised. It was found that the six-second isometric exercises produced increase in strength and endurance more rapidly than the DeLorme type of exercise. However, an additional increase in strength, and particularly in endurance, was found when six-second exercises were repeated several times a day. No contralateral effects were observed. The results were confirmed by a direct recording of the muscle contractions produced by electrical stimuli prior to and after training. Repeated brief isometric exercises were applied to patients with lower motor neuron lesions, with satisfactory results. The significance of these findings is discussed.

The Diaphragm and Intercostal Muscles during Expiration.

A. J. MURPHY *et alii* (*Arch. phys. Med.*, August, 1959) have continued their studies on the patterns of action of the diaphragm and intercostal muscles during respiration, dealing this time with expiration. They used a multiple-channel electromyograph with needle electrodes, and their findings were as follows. Activity in the diaphragm consistently continued from inspiration into passive expiration; the duration of carry-over activity was directly related to the size of the breath. Intercostal activity frequently continued into passive expiration; this carry-over activity was more likely to occur in the lower than in the upper intercostal muscles. The duration of carry-over activity in passive expiration was longest in the diaphragm, and decreased sequentially from the eleventh intercostal muscle upward. No activity could be found in the diaphragm during forced expiration. Intercostal activity was almost always present during forced expiration. The likelihood that intercostal activity would begin at the onset of forced expiration was inversely related to the volume of air in the lungs at the end of inspiration. When intercostal activity began after the onset of forced expiration, it was sequentially recruited from the eleventh intercostal upward. The hypothesis is made that during passive expiration the diaphragm and intercostal muscles exert a braking force against the elastic forces of the lung.

British Medical Association.

NEW SOUTH WALES BRANCH: SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 25, 1959, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. MUNRO S. ALEXANDER, the President, in the chair.

Complications of Operations.

DR. W. H. NEILD read a paper entitled "Post-Operative Complications" (see page 833).

DR. A. S. PATON read a paper entitled "Post-Operative Complications" (see page 835).

DR. K. S. JONES said that Dr. Neild had raised a number of problems, particularly the question of choleperitoneum. He asked Dr. Neild whether it was his policy always to provide an extra drainage tube, quite apart from any tube going into the common duct. If that was adequately placed, there would be external evidence of biliary drainage, and there would be no worry about whether choleperitoneum existed or not. Dr. Jones went on to say that, in dealing with post-operative intestinal obstruction, Dr. Neild had used the term paralytic ileus; he himself considered it a very vague term. In any case in which he had operated for intestinal obstruction, he had always found plastic peritonitis, not paralytic ileus.

DR. A. C. THOMAS, referring to Dr. Neild's statements about paralytic ileus, etc., said that the most common complication that he had ever had to deal with was acute dilatation of the stomach. On many occasions a colleague had telephoned to him, and always the clinical history was similar; five days previously an ordinary appendicectomy had been performed, and now it seemed that the patient was dying of peritonitis. When he examined the patient, the condition would be typical acute dilatation of the stomach; it was easily cured if treated early by the introduction of a tube and intravenous therapy. It was one of the commonest complications of abdominal surgery. Choleperitoneum was a condition that had to be looked for. Dr. Thomas said that for some years he would not use drainage in cholecystectomies. However, a few years back he had had an assistant who had been working as assistant to one of the leading London surgeons. At every cholecystectomy, during the suturing, he would remind Dr. Thomas that "So-and-so" always used drainage in similar cases. For conscience's sake, he himself began to use drainage, and was very pleased with the result. On one occasion the operation was dry, and there was a dry operative field, and the next day copious bile was draining from the tube. From then on he had always used drainage after cholecystectomy. Every now and again he was particularly pleased, because there would be copious drainage of bile which would certainly have caused much trouble if it had been allowed to remain inside. In every case drainage had ceased in three or four days, and the patient's convalescence had not been impaired. Dr. Thomas went on to say that Dr. Neild had gone through the whole gamut of post-operative complications; all had experienced them, and all had come to their own conclusions about the best way of preventing them. Some could be prevented. Post-operative hæmorrhage, for example, could be prevented if the vessels were tied as the operation proceeded. One surgeon had said that the way to treat it was with artery forceps and not blood transfusion; there was much in that. Present-day surgeons were perhaps not quite so careful with hæmostasis as the surgeons of the past. In his early days, one of the senior surgeons with whom he was associated never took less than an hour to perform an appendicectomy or a herniorrhaphy; he meticulously picked up every little vessel. Referring to Dr. Paton's remarks about post-operative vomiting, Dr. Thomas said that it was one of the things for which the anæsthetist was blamed at the present time. He thought that the surgeon must take some of the blame in abdominal operations; tender handling of the viscera was an aid to the good post-operative condition of the patient.

DR. S. V. MARSHALL expressed his appreciation of the fact that the surgeons had not blamed the anæsthetists for all the complications. He said that he had been interested in Dr. Paton's detailed analysis of the factors that could contribute to post-operative vomiting. With modern relaxants, there was a tendency to neglect adequate ventilation. Patients were thus allowed to accumulate carbon dioxide, which was a poison when it rose beyond its normal range of action. That also occurred when relaxant drugs were not suitably reversed. Such patients developed hypercapnia and hypoxia, the latter being a potent cause of post-operative vomiting. Respiration should be adequately restored before the patient passed out of the care of the anæsthetist. In his student days, he had been taught that carbon dioxide was a good thing, and that one could not have too much of it. Now it was known to be almost equal to hypoxia in its bad effects. Dr. Paton had mentioned how patients woke up, felt hungry, were given a meal and promptly vomited. Dr. Marshall said that they often felt thirsty and were given, for example, a cup of tea, which they gulped down; either a laryngospasm followed or they vomited, and the stage was then set for pulmonary inundation. Dr. Marshall thought that the complications mentioned by Dr. Paton should be remembered, and that an attempt should be made to avoid them.

DR. W. L. CALOV said that during the meeting the thing that had struck him forcibly, as on many occasions, was the high incidence of wound infections. He had had the experience of surgery in New Guinea in his early days, having had to perform most of the common operations; no doubt some had done so, but he could not remember any wounds at all breaking down. The operating theatre was whitewashed inside, the floor was of very rough concrete with channels in it, doors were open everywhere because of the heat, flakes of whitewash were peeling off the ceiling. The preparations for operation were very poor, and sweat streamed off the surgeon and into the wound. He had even seen flies drop down and fall into it. The wounds used to heal. Even after amputation of a leg for tropical sloughing phagedena, necessary to save the patient's life, the wound did not break down. Dr. Calov went on to say that a few years back he had again visited New Guinea, and with Dr. John Gunther had had to undertake the immediate surgical care of a woman who had fallen foul of her husband. She had gone off with another man, and the husband had attacked her with a large steel knife. One hand was hanging by a few tendons, and the knife had cut through her mouth and lower jaw, slicing off three of her incisor teeth. Dr. Calov said that Dr. Gunther had attended to the hand, while he had stitched up her mouth and jaw. Sterilization was negligible. The patient had been sent by air to a properly equipped hospital. She returned later, with even her teeth still there. The wounds had healed perfectly, with no sign of infection.

DR. T. E. WILSON said that the Section of Surgery had asked Dr. Neild to come down from Newcastle to take part in the combined meeting, as it was thought that, from his experience, he would be able to discuss the avoidance of post-operative complications. Dr. Wilson said that he just mentioned that fact, lest it be thought that the invitation implied that Dr. Neild had had more experience with the management of established complications than most surgeons. Dr. Wilson said that he agreed with Dr. Neild that only the minority of complications encountered were inevitable. In most cases they resulted from technical defects which one could discern on looking back, and which might have been avoided. If wound infection occurred, it could be due to a variety of technical faults, such as incomplete hæmostasis, unnecessary trauma to the tissues, failure to obliterate dead spaces in the wound, sloughing of the edges of deeper layers of the wound due to too tight suturing, the tying off of large pieces of tissue in the ligatures, and the use of a large amount of catgut in the wound. On the subject of non-absorbable sutures, Dr. Wilson referred to the advantages of cotton and stainless steel wire as suture materials. He asked Dr. Neild what were his views on the use of lumbar sympathetic block in the management of deep venous thrombosis of the leg; he himself had been impressed by the way in which the swelling of the tissues sometimes subsided after such an injection, which thus prevented the development of a "white leg".

DR. B. J. POLLARD, referring to wound infections, said that it appeared to be a matter of fairly common observation that in small private hospitals, where sterilization techniques were doubtful and the operating theatre linen could be seen through, wounds seemed to do better than in large institutions. He did not think it had anything to do with antibiotic-resistant strains; the same personnel operated at both institutions. Dr. Pollard asked Dr. Neild whether that had been his experience. It seemed that wound infection was not a problem in small

hospitals. Dr. Pollard then referred to what Dr. Paton had suggested as a test to establish the cause of hypopnœa, to determine whether the patient had received an overdosage of a curariform drug or had an idiosyncrasy to it—namely, the giving of a small dose of "Flaxedil". Dr. Pollard said he thought that was a rather dangerous thing to do, because if a relaxant was the cause, such a procedure would make things worse. There were other ways of determining the cause.

Dr. Neild, in reply to Dr. Jones, said that he used drainage after cholecystectomy as a routine procedure, and removed the drain only when there was a negligible amount of discharge. He did not think it mattered whether the drain was a tube or corrugated rubber, though the latter caused less pain and permitted the patient to breathe more freely. Referring to the drainage of bile through the tube, Dr. Neild said that he could recall three cases in which, after an operation which he had thought well performed, there was a profuse drainage, which ceased spontaneously. He could not see any breach in the biliary tree. The explanation might sometimes be a congenital abnormality; in 14·5% of cases an accessory right hepatic duct was present and drainage to it could easily go unnoticed. In choleperitoneum the behaviour of the tube was always worth investigating. In two cases in Mackenzie's series, there was no drainage of bile from the tube at all; in one of his own the same thing had occurred, but there were significant amounts in the peritoneal cavity. If a T tube was used to drain the common bile duct, it was wise to bring it out through a different wound from that used for the drainage tube in the sub-hepatic pouch. The reason was shown in a case of which he had experience; the two tubes had been brought out through the one incision, and when one was being extracted on the fourth day, both came out; there was much alarm, but all ended well. Dr. Neild then referred to what Dr. Jones had said about paralytic ileus. He said that he thought there was such an entity. Occasionally cases had been recorded in which procedures not connected with the abdomen at all, such as the mere application of a plaster spica, had resulted in dilatation of the small bowel and vomiting. Dr. Thomas had said that he considered acute dilatation of the stomach to be much more common than paralytic ileus. Dr. Neild said that he was interested to hear that, but it did not agree with his experience. So-called plastic peritonitis had been considerably more common. He had had experience of one case, after a gall-bladder operation. With regard to post-operative hæmorrhage, Dr. Neild said that there was much truth in Dr. Thomas's comment that the best hæmostatic was artery forceps. In reply to Dr. Calov, Dr. Neild said that wound sepsis was a rather sorry story at present. It seemed to be a problem of doctors' own making; by their excessive use of antibiotics they had created a bacterial Colossus. A vast number of hospital workers carried staphylococcal infections in their nares, and deposited the organisms into the wound. As Dr. Pollard had said, it was true that the smaller hospitals had a lower infection rate. Lawson Tait had found that out in his day; when a large new hospital was being built in Birmingham, he had written to the governors and advised them to pull it down. Dr. Neild said that he had worked in a few small hospitals in Newcastle; certainly the operations he had performed there were smaller procedures, but the incidence of wound sepsis was very low. In reply to Dr. Wilson's question about lumbar sympathetic block, Dr. Neild said that he had seen it used on a few occasions, but had not seen any dramatic result. The procedure had first been suggested by Leriche. In thrombophlebitis there was so little to fall back on that there was nothing to lose by trying it.

Dr. Paton, in reply to Dr. Pollard, said that he agreed that the test was a dangerous one, but giving an anæsthetic was also dangerous. He did not recommend the test; but it was a method of distinguishing between two types of block. The dose was 10 mg., which was very small and would not do much harm. The object of the test was to distinguish between depolarizing and competitive block; there would be no effect on the former. Dr. Neild had mentioned noradrenaline. Dr. Paton agreed with him that its use was a last step before the post-mortem examination. The patient's cardio-vascular system had taken a severe pounding, and the patient was usually in a state of irreversible shock. The use of noradrenaline only made it more irreversible.

Dr. Alexander, from the chair, thanked the speakers for their papers, and those who had joined in the discussion.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

PUBLIC HEALTH IN HOBART.[1]

[From the *Australasian Medical Gazette*, January 20, 1897.]

THE annual report of the Health Officer for Hobart has just been published. Dr. Sprott calculates the death-rate of Hobart as 12·97 per 1000 of population. With the exception of typhoid fever and diphtheria the city has been free from infectious diseases. In the early part of the year influenza and whooping cough were prevalent, with a few fatal cases. There were four cases of scarlet fever and one of measles notified but no fatal results. The death-rate from diphtheria is higher than usual, but this is owing to the fact that an epidemic broke out in the month of February last. The infection was spread through milk supplied by one of the country dairies. As soon as the city authorities were aware of the source of the infection, the necessary steps were taken to prohibit the sale of the tainted milk. There were 45 cases notified during the year, and 29 of these were traceable to the above source of the infection. There were nine deaths. During the early part of the year typhoid was prevalent, but for the past few months it has been wholly absent. There were 67 cases notified. There were 11 deaths. Of the other preventible diseases phthisis and other forms of tubercular disease contribute very largely towards the death-rate. It is to be hoped, now that consumption has been recognized as an infectious disease, that the death-rate may be reduced. The by-law introduced by the Board of Health prohibiting people expectorating on the streets and footpaths may do something towards this reduction. It is a matter of regret that Hobart and the colony generally are practically without vaccination. The compulsory clauses of the Vaccination Act have not been enforced for the past seven years.

Correspondence.

SOME ASPECTS OF ALCOHOL IN BODY FLUIDS.

SIR: The two articles by McCallum *et alii* (MED. J. AUST., August 8, 1959), "Some Aspects of Alcohol in Body Fluids", have received considerable publicity derogatory to the value of blood alcohol estimations for "driving under the influence" cases. On the evidence of the findings, certain of the conclusions of the authors cannot go unchallenged. The nature of their experiments were not comparable to what would obtain in actual practice. They also confused two fundamental issues, "driving under the influence" and "drunken driving".

Their work added nothing new to the problem, and nothing which had not been carefully weighed and considered by the Medico-Legal Committee. Their papers did not emphasize that the report of the Commonwealth Medico-Legal Committee established by the Australian Transport Advisory Council provided in its draft regulations only a guide for the analyst in his assessment of the blood alcohol level where a "back calculation" was necessary, and that the analyst was free to make his assessment from all the data available. The authors stated that the Special Committee of the British Medical Association in England said "that the clinical examination in most cases must remain the determining factor in arriving at a final diagnosis and the laboratory diagnosis must be considered as an adjuvant". This is not taken from the original report of the committee, but as quoted from Glaister.

A reference to the actual report is relevant. It states on page 7:

> It should be emphasized that a person need not be grossly incoordinated in his movements and "drunk" in the popular sense before he can be found to have committed an offence within the meaning of the words of the *Road Traffic Act*.

[1] From the original in the Mitchell Library, Sydney.

And on page 22:

> An examining practitioner should base his opinion in the first instance solely on his clinical findings, modified subsequently, if necessary, in the light of the results of any laboratory tests.

It must be emphasized that the Medico-Legal Committee's recommendations were for assistance to the court in determining intoxication, and the committee was unanimous that the evidence of the blood test was only to supplement verbal evidence, and not to be used as conclusive evidence either of sobriety or insobriety.

Clause 10 of the report is specific in this matter:

> Nothing in this Act shall be construed as limiting the introduction of any other competent evidence bearing on the question whether any person was or was not under the influence of intoxicating liquor, whether such evidence is in addition to or independent of any evidence provided for in this Act.

Again in Clause 3:

> Provided nothing in this section shall limit the right of either party to require such analyst to appear in any proceedings and give oral evidence on oath.

It is obvious from these extracts from the Draft Act that the Medico-Legal Committee took the greatest care that no injustice could be done to the defendant, and that the analyst's findings and assessments would be subject to scrutiny and cross-examination before being accepted.

It is now necessary to define "driving under the influence" as an entity distinct from, but inclusive of, degrees of intoxication involving incapacity. Numerous judgements both in Australia and in other countries were examined, and for its deliberation the Committee accepted the definition of the Court of Arizona, U.S.A.:

> The expression . . . "under the influence" covers not only all the well known and easily recognised conditions and degrees of intoxication, but any abnormal mental or physical condition which is the result of indulging in any degree in intoxicating liquors, and which tends to deprive him of that clearness of intellect and control of himself which he would otherwise possess. If the "ability" of the driver of an automobile has been lessened in the slightest degree by the use of intoxicating liquor, then the driver is deemed to be under the influence of intoxicating liquor. The mere fact that the driver has taken a drink does not place him under the ban of the Statute unless such drink has some influence upon him, lessening in some degree his "ability" to handle the said automobile.

Some authorities confine their attention to degrees of incapacity associated with obvious clinical evidence of the third stage of drunkenness—i.e., when the Thalamus and Cerebellum are obviously affected—and it is these authors who use figures as high as 0·20 W/V%.

All authors who were concerned primarily with determining the blood alcohol level at which there was a demonstrable impairment of mental and physical exercises have concluded that the range is from 0·03 to 0·07 W/V%, and that the vast majority of human beings have impairment of their faculties required for the proper handling of a car at a concentration of 0·05 W/V% or lower.

The classical studies of Bjerver and Goldberg lead them to the conclusion "the threshold of impairment of driving ability in expert drivers accustomed to consuming moderate quantities of alcohol is an alcoholic concentration of 0·035 to 0·04 W/V%".

During 1958 three separate investigations—Cohen in Manchester, Drew in London and Loomis and West in Seattle, U.S.A.—each placed the level blood alcohol at 0·05 W/V% at which their experimental subjects showed impairment.

In the U.S.A. there has been a significant swing away from the original line of demarcation of 0·15 W/V% to 0·05 W/V%, and up to 1956 no less than 27 States had adopted 0·05 W/V% as the line of demarcation. In Norway, 0·05 W/V% is presumptive evidence of guilt and no other evidence is required. It is worthy of reflection that Norway has maintained a road mortality rate of four per 100,000 of the population, whereas in several Australian States the rate is of the order of 24 per 100,000 of the population.

With this background, it is relevant to analyse McCallum's findings to see in how many cases an injustice would possibly have been done by accepting the findings of the maximum blood alcohol levels. In their Table I, result of Experiment A (page 174), there are 56 subjects; of these at the end of drinking 26 had blood alcohols at or above 0·1 W/V% and 20 at or above 0·05 W/V%, and these can be definitely excluded from further analysis. This must be clearly understood, that from a legal and practical point of view that the offence is not to be related in degree to the blood alcohol level once the line of demarcation has been exceeded. Once impairment of his faculties obtains, a driver is just as big a potential danger irrespective of the actual blood alcohol level, and many of the most serious accidents occur long before the driver is clinically drunk.

Of the 10 whose blood alcohol was below 0·05 W/V% at the end of drinking, one subject did not reach 0·05 W/V%. Of the remaining nine, at the end of drinking, four were above 0·04 W/V%, and the maxima reached were respectively as shown in Table A.

TABLE A.

Blood Alcohol Level.

At the End of Drinking.	Maximum.	Time. (Minutes.)
0·042	0·099	60
0·048	0·070	60
0·048	0·107	90
0·045	0·078	75

From a practical point of view, it is obvious that by the time such subjects left a hotel, reached their cars and drove off, sufficient time would have elapsed for their blood alcohol level to have risen above 0·05 W/V%.

Of the remaining five cases, Subject 4, who drank 28 oz. of beer in 34 minutes, reached a maximum of 0·052 at 60 minutes later. This, from the legal point of view, would obviously be a borderline case.

Subject 17 drank 56 oz. of beer in 30 minutes, his blood alcohol level rose from 0·03 to 0·082 in 90 minutes.

Case 27 drank 56 oz. of beer in 30 minutes, his blood alcohol rose from 0·037 to 0·071 in 75 minutes.

Case 37 drank 56 oz. of beer in 35 minutes, his blood alcohol rose from 0·033 to 0·093 in 85 minutes.

Case 41 drank 56 oz. of beer in 20 minutes, his blood alcohol rose from 0·017 to 0·075 in 110 minutes.

The data which are lacking in these four cases is the time at which a level of 0·05 W/V% was reached, and unless these data are available it is not relevant to consider them further, as from the practical point of view these people were not under the influence at the time the initial blood sample was taken, and the chances of them having an accident before they did reach a significant blood alcohol level would be no greater than that of a sober population; but as their blood alcohol level was rising and they had drunk enough to bring them under the influence, they would be prone to be rapidly affected. It is well known that it is a rapidly rising blood alcohol which has the maximum effect on the brain. Once sufficient time had elapsed from the cessation of drinking to the time they were under the influence, then their blood alcohol level would have been significantly closer to the maximum they reached, so that the disparity found by McCallum would not have occurred under practical drinking conditions.

The Medico-Legal Committee's chief aim was to establish a line of demarcation for safe driving, and its primary purpose was not the punishment of wrong-doers, but the education of the public of the dangers of driving under the influence.

The rapidity at which the alcohol was drunk (the maximal fastest rate possible for the individual) was in itself unnatural behaviour for a social drinker, and weighed the experimental findings unfairly in favour of the disparity between the maximum and minimum blood alcohol levels. In other words, the experiments cannot be considered as a fair analysis of the postulated shortcomings of the validity of blood tests under normal drinking habits.

Again, one factor which must not be overlooked is that the very object of the experiment would in many cases have produced physiological disturbances in some people, associated with changes in the stomach, intestines and vascular supply to these organs which would inhibit the rate of alcohol absorption. The authors themselves acknowledge that their experiments were unphysiological, and state: "In particular we wish to acknowledge our debt to those several subjects who so willingly endured the

exacting and sometimes harsh conditions of the experiments."

Finally, the experiments prove to an exceptional degree the main contention of all who want safety on the roads—namely, swilling is the most pernicious and dangerous way of consuming alcohol.

The authors' discussion would seem to imply that the different rates of absorption and difference in the rates of alcohol drunk to blood alcohol concentrations were of moment in the over-all problem, but this is not so; the great value of the blood alcohol level is the figure *per se*. The committee was fully aware that it cannot and should not be related to the alcohol consumed. That it is so related at times is only because of the difficulty that the Court may find in associating it with the minimum quantity of alcohol consumed. This is only a transitory phase, and it will soon be accepted that the level of the blood alcohol should be no more related to alcohol consumption than is the sugar tolerance curve to the amount of sugar ingested.

The committee was also aware of the fact that weight alone is not the determining factor in difference in blood alcohol level for a given dose. A short fat man has a different total water content to a tall man of equal weight.

The authors' findings are a valuable piece of research; but some of the deductions drawn from the experimental research are unacceptable, especially in relation to the statement that a blood test which is not taken immediately after a charge has been laid is valueless.

Conclusions.

1. McCallum and his co-workers' experiments were in many instances unphysiological and cannot be related to normal drinking habits.

2. The results of their experiments in no way invalidate blood tests, and there is no evidence of substance which invalidates the assessment of the blood alcohol level from the level of alcohol in the blood taken subsequent to the alleged offence.

3. Comprehensive safeguards to protect the defendant under all practical drinking conditions were written into the recommendations of the Medico-Legal Committee. The findings of McCallum and co-workers do not prove these safeguards to be inadequate.

4. The authors do not accurately report the recommendations of the Special Committee of the British Medical Association of England in relation to the diagnostic value to be placed on blood tests.

5. The findings of McCallum and co-workers highlight the potential danger to others when drivers drink large quantities of alcohol on an empty stomach. The rapidity at which the blood alcohol level rose, and the height attained during the drinking period in a substantial majority of their subjects, emphasize anew the dangers of swilling.

6. The Medico-Legal Committee's recommendations emphasize that blood tests are to be used only to supplement witness evidence, but it stresses that blood tests are scientific, accurate and free from bias for or against the accused.

7. Through its deterrent value, the legal recognition of blood tests strictly enforced would make a substantial reduction in deaths and casualties on the road.

Yours, etc.,

F. S. HANSMAN,
A. E. STONHAM.

Sydney.
Undated.

THE DANGERS OF MOTOR LAWN MOWERS.

SIR: Most suburban practitioners will agree with a suggestion that the designers of motor lawn-mowing machines should try to minimize the dangers associated with their use.

It has been my experience that two types of injury arise: (i) lacerations from the rotating blades; (ii) puncture wounds from stones thrown up by the blades. In the past four years I have treated 36 patients for injuries of this type. (a) Sixteen patients with multiple lacerations of feet, usually the great or little toes of the left foot. The wounds have been quite extensive, and the inconvenience to the patients has been considerable. (b) Five patients with stones embedded in the muscles of the lower leg. These were easily removed, but the wounds were extensive. (c) One patient, age 9, with a severed Achilles tendon, requiring hospitalization and many weeks of treatment. (d) Thirteen patients with lacerations to their fingers, usually (10 out of 13) the index finger and thumb of the right hand. (e) One patient, a child, two and a half years, who suffered a hæmorrhage into the anterior chamber of the eye and a tear in the iris from a stone thrown at high speed. I think the accident rate from this type of injury in the metropolitan area would be quite high, and that the designers of these machines would need little ingenuity to alter the machines so that the safety factor is increased.

Yours, etc.,

A. H. SHEEHAN.

42 Burwood Road,
Concord.
November 23, 1959.

MORBIDITY RECORDING IN A SMALL COUNTRY PRACTICE.

SIR: As stated in my original manuscript, I am indebted to Dr. R. J. F. H. Pinsent, Chairman of the Research Committee of the Council of the College of General Practitioners, for the following passages in my paper "Morbidity Recording in a Small Country Practice": "work has been done by individuals (McGregor, Fry, Watts and Horder), each independently using his own method of classification and analysis. An unfortunate result of this is that figures independently collected, were not always comparable". In the process of editing this was attributed to the "report of the Medical Research Council (1944)". This is not the case, as the work referred to was done later, and the communication of Dr. Pinsent to me was dated June 6, 1957. For the same reason the analysis of Logan (1952) does not refer to the survey of 108 practices, which was only published in December, 1958 (as Dr. Elmerl, of the Research Committee of the College has been kind enough to point out to me). This reference to Logan's paper also was the result of editing. When reading the proof, I did not notice this discrepancy, and to avoid embarrassment of the authors of this splendid survey I should like to publish this correction.

Yours, etc.,

HANNS PACY.

Tea Gardens, N.S.W.
July 9, 1959.

[These errors were due to a misunderstanding and are regretted.—EDITOR.]

REPORT OF A CASE OF DIFFUSE INTERSTITIAL FIBROSIS OF THE LUNGS (HAMMAN-RICH SYNDROME) SUCCESSFULLY CONTROLLED BY PREDNISOLONE.

SIR: The discussion (MED. J. AUST., August 22 and October 31) following Dr. Bennett's paper on diffuse interstitial pulmonary fibrosis (MED. J. AUST., July 18) stems partly from the hazard of drawing general conclusions on the basis of a single case. It seems appropriate to summarize the progress of a small series of 11 patients.

I have been closely associated with the management of four patients with this condition for periods of one to two and a half years. Two, including the only male, are dead after showing no response to full doses of cortisone or prednisone; one is alive, but also has shown no improvement. In the fourth patient's case treatment was begun when the condition deteriorated after several months of observation; a dramatic improvement in exercise tolerance, not reflected by any change in ventilatory capacity, was maintained for about a year. Further deterioration in exercise tolerance occurred recently, and an increase in the maintenance dose of prednisone has again been associated with a remarkable response. At her worst, before cortisone, she could barely walk a few yards because of dyspnœa; when seen today she had twice in the past week walked over a mile on the flat without a rest. This patient's exercise tolerance always varied a little from day to day, suggesting reversible bronchoconstriction; but there has never been any audible wheeze, nor was the forced expiratory volume at half a second increased after a potent bronchodilator aerosol. In addition, the large area over which inspiratory crepitations were originally heard decreased by more than two-thirds after prednisone was

given, suggesting perhaps that the site of action was alveolar rather than bronchial or bronchiolar.

Seven further patients (two male) have been referred to me for pulmonary function studies and have been examined on one or more occasions. One severely ill woman responded dramatically to cortisone but has, I believe, relapsed since in spite of maintenance therapy. Three given cortisone or prednisone have shown no improvement. The remaining three, diagnosed recently, have not as yet been given steroids. One of these, with typical clinical features and clear evidence of alveolo-capillary block, has noted definite spontaneous improvement in exercise tolerance over several months preceding diagnosis; no lung biopsy has been done.

In only one patient has an isoprenaline aerosol produced significant improvement in the half-second or one-second forced expiratory volume. The increase was small (about 15%), and neither the patient nor the test results improved after prednisone. Only one of the two patients who did improve clinically after cortisone was effectively tested in this way, but no increase occurred after isoprenaline. In spite of these negative findings, reversible bronchoconstriction could explain the increase in ventilatory capacity after treatment in Dr. Allen's case. This possibility would have been confirmed or denied if the tests of ventilatory capacity had been done before and after a bronchodilator aerosol on each occasion. This procedure is essential to the effective interpretation of these tests; an obstructive element in association with a restrictive defect is otherwise easily overlooked. However, in spite of my failure to demonstrate significant reversible bronchoconstriction by this reasonably sensitive method, the hypothesis of Dr. Read and Dr. Colebatch remains a possibility; there is evidence that cortisone produces some bronchodilatation by a mechanism not available to isoprenaline, or it may act, as your correspondents have agreed, by altering intrapulmonary mechanics.

In summary, therefore, I do not feel that Dr. Bennett's favourable experience with cortisone in his case accurately reflects the therapeutic situation in this distressing but fascinating disorder, nor do my observations, admittedly limited, support Dr. Read and Dr. Colebatch's suggestion as to the mode of action of cortisone in those cases in which it is beneficial.

I am indebted to the physicians who have kindly referred the latter group of patients for study, and to Dr. Murray Maxwell for information on the recent progress of two of the patients mentioned.

Yours, etc.,

BRYAN GANDEVIA.
University of Melbourne Department of Medicine,
Royal Melbourne Hospital,
Parkville, Victoria.
November 6, 1959.

DRUG HOUSE REPRESENTATIVES.

SIR: I would like to support Dr. Molesworth's remarks in the Journal of October 31, 1959. In practice here, I am compelled to see the representatives of the drug houses by appointment only. My main trouble is that these gentlemen appear to hunt in packs. Five may come along in one week, all stressing the claims of five different brands of the same basic product. Visits could be spaced to the better advantage of all concerned.

I think Dr. Molesworth could also have drawn attention to the vast "literary" output of the same firms. It overflows our mail-boxes. We admit that some of it is of value, but no doctor could make a perusal of the lot, unless he devoted a couple of mornings a week to it.

I suppose it is too much to ask for a simpler terminology. Also, I look forward to the appearance of the genius who can sort out the endless variety of glandular products now flooding the market. He might even discover some symptom complex which these wonder products cannot cure.

Yours, etc.,

J. J. MCINTOSH.
Mareeba,
North Queensland.
November 11, 1959.

SIR: I have read Dr. James Molesworth's letter in the issue of October 31, 1959, with interest.

I have instructed my secretary to inform all drug house representatives that I will interview them at 9 a.m. on Wednesdays and at no other time. Very few of them have failed to keep appointments, and most of them, knowing my specialty, confine themselves to what is likely to be of interest to me. They are reasonably brief, courteous and helpful, and do not talk about vitamins. They are welcome to make periodical visits. There are the odd few who are ignorant and irritating gasbags, but on the whole the system works amicably and well.

My only grievance against the drug manufacturers is that in some cases their blarney departments persist in flooding my private residence with their mendacious literature.

Yours, etc.,

F. V. STONHAM.
2 Collins Street,
Melbourne.
Undated.

THE TUBERCULOSIS CONTROL CAMPAIGN.

SIR: Last year, in New South Wales, five times as many people died from tuberculosis as died from all other notifiable infectious diseases put together. Last year also, 1399 new cases of tuberculosis were notified. Many were of people who had little or no suspicion that they had the disease. In other words, the disease was found in well over 1000 members of the community who quite unwittingly could have been, and some were no doubt, spreading the infection to other members of their family, or to their friends or workmates.

Unfortunately, despite this evidence that tuberculosis is still a major problem, there is a great deal of apathy towards the Tuberculosis Control Campaign. We all know far too well that people generally only become really concerned about their health when they lose it. The chest X-ray is, without doubt, still the best single means of detecting pulmonary tuberculosis in the apparently healthy. For this reason the National Association for the Prevention of Tuberculosis in Australia asks for continued help in the Commonwealth-wide fight against this disease.

I wish to bring to the notice of all medical practitioners the desirability of advising the making of a chest X-ray film, not only in those cases who present themselves with chest symptoms, but also in those who complain of indifferent health over a period. It is well to remember also that many other conditions, including bronchogenic carcinoma, have been revealed by what might be termed routine X-ray.

It can be said that all medical practitioners must play an ever-increasing part in this campaign for it to be completely successful. I ask, therefore, that they will combat in every way possible the growing complacent attitude towards tuberculosis and its control.

Yours, etc.,

MARSHALL ANDREW,
Chairman, Educational Sub-Committee.
National Association for the Prevention of Tuberculosis in Australia,
59 Holterman Street,
Crow's Nest, N.S.W.
Undated.

AN IMPROVED METHOD OF PROSTATECTOMY.

SIR: In your Journal of October 10, 1959, I was interested to read Mr. J. M. Foreman's concise criticism of the article by Mr. Bruce Pearson entitled "An Improved Method of Prostatectomy".

Generally, I quite agree with Mr. Foreman's observations concerning the virtues of the Millin prostatectomy, although 25 to 30 minutes seems a trifle brisk for a carefully performed hæmostatic retropubic prostatectomy. I find from my own experience, and from that of most of my teachers at St. Peter's Hospital for Stone, that a preoperative cystoscopy and retropubic prostatectomy, with careful attention to hæmostasis and capsular closure, combined with bilateral vasectomy, takes an average of 60 to 75

minutes. My impression is that rapidly performed cases where hæmostasis and capsular closure may be a little deficient, have an increased morbidity and lengthened stay in hospital, whereas the more leisurely performed retropubic operation is usually ready for discharge from seven to 12 days post-operatively.

The fact that different urologists sing the praises of one particular prostatectomy procedure suggests that, if well done, most methods are good and give excellent results. My colleagues at the Royal North Shore Hospital claim fine results with the Harris transvesical prostatectomy, and Mr. Bruce Pearson is happy with the transvesical modification that he employs. Dr. Elmer Belt, with whom I worked in Los Angeles, gets superb results with the perineal approach, while Dr. Roger Barnes of Los Angeles does almost all his prostatic operations by the perurethral route, with enviable results. Finally, in the last 10 years most British-trained urologists have swung to the Millin procedure in preference to the transvesical for their open cases.

Professor Willard E. Goodwin, of the UCLA Medical Center, surely sums up the situation correctly when he states that the modern, well-trained urologist should know how to do all these procedures adequately, because every patient is a different problem and there is no single operation that meets the requirements of all cases.

If a surgeon must press his method above all others, would it not be more objective for him to say: "The operation I do is the best—in my hands!"

Yours, etc.,

149 Macquarie Street,
Sydney.
October 27, 1959.

ROBERT D. SMITH.

Obituary.

JOHN KELLERMAN ADEY.

WE are indebted to Dr. J. Catarinich for the following account of the career of the late Dr. John Kellerman Adey.

John Kellerman Adey, affectionately known as "J.K." to his confrères, was born at Thornton, Somerset, England, on November 26, 1887, and came to Australia with his people when 14 years of age. He was educated at Melbourne Grammar School, and proceeding to the University of Melbourne, graduated in 1909 at the early age of 21.

After a period spent as resident medical officer at Warrnambool Hospital, Adey's attention was attracted to psychiatry, and he joined the Victorian Department of Mental Hygiene on October 1, 1911. He served in various mental hospitals as a junior officer, and after serving in the first World War was promoted senior medical officer. In August, 1919, he was transferred to Royal Park, where he came under the influence of Clarence Godfrey, a name and personality to remember. During these early years Adey's varied experience and common-sense application of what was good in his environment were gradually moulding and maturing him into a psychiatrist of decision and original ideas. In 1922 he was appointed medical superintendent of the Sunbury Mental Hospital, and very soon he set about enlarging the liberty of the patients. Hitherto, patients suffering from mental disorder were regarded much as prisoners, the illness being quite subsidiary to the maintenance of discipline throughout the institutions. Adey unlocked doors and established a system of parole which was rarely abused by his patients. It was during this period, also, that he and Ellery began the malarial therapy of neurosyphilis. Subsequently, this form of treatment was concentrated at Mont Park, where much better facilities were available.

In 1932 Adey took over control of Royal Park, and immediately installed open wards for patients acutely ill. As superintendent of Royal Park, he was at the same time appointed lecturer in psychiatry and very soon he was attracting attention by the acumen of his observations and the clarity of his statements. Until his retirement he remained one of the most popular of university teachers. But it was not only the students who were indebted to him. At Royal Park he established weekly clinical meetings, where all were welcome at the demonstrations and discussions—the highlight of which were the crisp sentences with which he summed up the cases presented and indicated the treatment and any further investigation he thought necessary.

In 1939 he became a member of the Faculty of Medicine, acting as the representative of the Victorian Government, and held the position until 1953. During this period he took a large part in the preliminaries which led to the establishment of the diploma of psychological medicine at the University of Melbourne. In between times he was acting as Deputy Director of Mental Hygiene during the absences of the Director. In April 1952 he was appointed Chief Medical Officer of the Department of Mental Hygiene, which office he held until his retirement. Intolerant of idleness, Adey immediately started practice as a consultant psychiatrist.

His academic activities were many. At the Australasian Medical Congress (B.M.A.) held in Adelaide in 1937, Adey was given the honour of presiding over the Section of Neurology and Psychiatry. He was a Foundation Fellow of

The Royal Australasian College of Physicians, and a Foundation Member of the Australasian Association of Psychiatrists, of which he was President in 1954. An active member of the Medico-Legal Society, he was also interested in the Section of Medical History of the Victorian Branch of the B.M.A.

Adey was proud of his association with the Royal Society of St. George, and at the time of his death he was the society's senior vice-president.

Adey served in both World Wars. He was on active service on Gallipoli and in France and Germany and at the end of the first war he was lieutenant-colonel in charge of the 3rd Casualty Clearing Station. In the second war he was in the Middle East as colonel in charge of 2/1 Australian General Hospital. He was mentioned in dispatches in both wars, and was honoured with the O.B.E. in 1917.

Some time before his death, Adey had an attack of coronary occlusion. He sent for his senior medical officer and advised him of the diagnosis. It was characteristic of the man that his ailment made no difference to his activities, and he was soon hard at work again. All through his life he possessed a restless energy which exhibited itself in everything he did, even in recreation. He played keenly bridge, tennis and golf. A firm disciplinarian, he was recognized by his staff as just in his decisions and a good friend in adversity. Eminently approachable, he was careful never to hurt anyone's susceptibilities. Always he carried his responsibilities unobtrusively, yet with dignity. He was

a friend to take to one's soul. Throughout his life, like the shadow of evening, he steadily increased in stature until the sunset came. His memory will be cherished by many.

MAJOR ARTHUR E. PRITZ writes: It is with a sense of high privilege that one approaches the task of writing a few notes on the military career of Colonel J. K. Adey, O.B.E., an officer of the Australian Army Medical Corps who had long and distinguished service.

Born on December 26, 1887, Adey was first commissioned in the 3rd Light Horse Field Ambulance in 1911, which must have been soon after his graduation in medicine at the University of Melbourne. There followed a trip abroad for post-graduate study, and one day in 1913 young Adey attended a military parade in Edinburgh. On that day he met the future Mrs. Adey, so thus early it is apparent that soldiering was to be an important part of his life.

He had not long been back in Australia when came the first World War. Adey was soon in uniform with the 6th Field Ambulance of the A.I.F. and embarked for overseas with the Second Division. It is interesting to recall some names in that unit. It sailed from Australia in H.M.T. *Ajana*, and after somewhat of a Cook's tour—visiting Colombo and Bombay, and coming back to Colombo—it arrived at ANZAC on September 4, 1915. The unit was commanded by Lieutenant-Colonel G. H. W. Hardy of Ballarat. A Section was commanded by Major Paul ("Chook") Dane, with Adey as his senior captain. B Section was commanded by Major J. E. M. Deakin of Adelaide. A senior non-commissioned officer was Sergeant Douglas Thomas, who was later returned to Australia to complete his medical studies. Captain E. R. Cordner, the founder of the famous football dynasty, who has supplied these details, thinks that he is now the sole survivor of the original officers.

Evacuated sick from the Peninsula, Adey again saw operational service with the ambulance in Egypt in early 1916 and was promoted major at Tel-el-Kebir in February of that year. There followed various appointments in England, France and Belgium. We find him promoted Lieutenant-Colonel to command the 3rd Australian Casualty Clearing Station on May 1, 1918, which appointment he held until the Armistice. His distinguished services with First Australian Imperial Force occasioned a mention in dispatches and the award of an O.B.E.

In 1936, with the revival of interest in Australia's defence forces, Lieutenant-Colonel Adey was back in harness commanding the 5th Casualty Clearing Station of the Commonwealth Military Forces. He continued with that unit until April 4, 1940, when he took over command of the newly-formed 2/1 Australian Casualty Clearing Station, of the Second Australian Imperial Force. This unit had been formed and partly trained by Lieutenant-Colonel F. K. Norris. On appointment of Norris to Assistant Director of Medical Services, 7th Division, Adey took over the unit just prior to its embarkation with the 17th Infantry Brigade in April, 1940. Thus he was commanding his third casualty clearing station, his second on active service, and was starting again where he left off in 1918.

Embarking for the Middle East in H.M.T. *Nuralia*, Adey laid about him vigorously to complete the training initiated by Norris. It was soon realized that here was a man who brooked no loafers and tolerated no fools. Also joining the unit at this time were eight sisters of the Australian Army Nursing Service, and Adey at once gave to Sister J. Hanna an important status in the hierarchy of the unit. *Nuralia* was a packed ship, and Lieutenant-Colonel A. H. Godfrey, Commanding Officer of the 2/6 Battalion, asked Adey to give a series of talks to men of his battalion and other units aboard. These addresses became a notable feature of the voyage. Each afternoon Adey paced the deck, talking to the men on military hygiene, venereal disease and, particularly, the role of the Australian Army Medical Corps in evacuation and treatment of casualties. His obvious knowledge of his subject, delivered with a down-to-earth method of speaking and a great sense of humour, made him a great success with the men, and had a profound effect on the morale of these young soldiers preparing themselves for active service.

In Palestine and Egypt the casualty clearing station opened small hospitals, and then on New Year's Eve, 1940, moved to Mersa Matruh, its first battle station. Here the Assistant Director of Medical Services, a British officer, was shocked to learn that Adey had brought his nursing sisters, and ordered their immediate return to base. These instructions Adey ignored, and he proceeded to make proper arrangements for their comfort. Within three days the unit was handling battle casualties from Bardia, and the Commanding Officer kept his sisters. Here Adey's true worth was realized by his younger officers. His great knowledge of the army machine, his coolness and power of instant decision impressed all ranks. So did his great care of his patients. He at once laid down the dictum that casualties must be fed before they were doctored. It was a standing order that at any hour of the night or day, on arrival of a convoy, the cooks were to be roused and a hot meal served within the hour. One had to be in a ward to realize the effect produced by roast beef and potatoes on men who might have spent twenty-four hours or more in an ambulance on the rough desert tracks. Second only to this as an uplift was their first sight of an army sister.

Towards the end of the Libyan campaign, Adey was posted to command 2/1 Australian General Hospital at Gaza Ridge, with promotion to colonel in January, 1941. This command he held until his discharge on medical grounds in September, 1943, when the unit was stationed in Western Australia. With the Australian General Hospital he established a high reputation as an administrator. With the establishment of Australian Imperial Force Headquarters in the Middle East, and the expansion of the force, the Director of Medical Services appointed specialist advisers. Colonel Adey became adviser on psychiatry, and made many visits to medical units in this capacity. His advice was invaluable during the difficult period of rehabilitating the force after Greece and Crete. For a period he was a full-time staff officer at Australian Imperial Force Headquarters, combining the duties of adviser on psychiatry with those of the "A" staff officer, dealing with personnel and reviewing medical boards. In 1942 came his second mention in dispatches for his second war. One might also mention that in 1941 Adey's son was also on active service in the Middle East.

This recital of service would not be complete without reference to some facets of Adey's character which made him a distinctive figure in the 2nd Australian Imperial Force. A man of few words, probably a shy man, he was short to the point of curtness in conversation, but a most successful lecturer to other ranks. Here his humour and his fatherly approach earned the affection of his audience. He probably never held an officer's conference, but took all decisions himself, instantly and very definitely. His decision was inflexible and permanent, and he who acted upon it immediately had always the full support of his commanding officer. In addition, he delegated responsibility to the fullest and obtained loyalty by so doing. He had little time for non-essentials, as evidenced by his personal dress, which was a byword in the division. The despair of his batman, he just could not keep his shirt within his belt. The sight of the commanding officer in his "Bombay bloomers" was something that had to be seen to be believed. It is narrated that on one occasion a visiting British general officer, on inspecting the commanding officer, refused to inspect the unit. This would cause Adey no more than an inward chuckle. His orderly rooms were models of brevity, justice being dispensed by intuition with little concern for evidence. The recipient usually departed satisfied, if the sergeant-major did not.

Adey held very high the status of the Australian Army Medical Corps. He refused to allow the medical services to be used as a convenience for moving on a man who was not *persona grata* with his commanding officer. Medical vehicles were for the transport of patients and no one else. His unit was never short of supplies or equipment, and the care of his men was a prime consideration. It was very obvious that commanders in other arms of the force regarded him with great respect. In his own unit, his fame increased after he had left it. Even in these post-war days, when men are discussing some unfortunate happening, it is common to hear the remark: "That would not have happened in Co. Adey's day." This, the writer submits, is high tribute to his success as a commanding officer.

MONTEFIORE DAVID SILBERBERG.

WE are indebted to Dr. Clive H. Fitts for the following account of the career of the late Dr. Montefiore David Silberberg.

Dr. M. D. Silberberg, Consultant Physician to the Alfred Hospital, Melbourne, died on June 24, 1959, at the age of 76 years. He was born on August 27, 1882, at Branxholme, Victoria, the youngest son of the storekeeper of that small country town. He was educated at the local State school and at Wesley College, Melbourne, and entered Queen's College at the University of Melbourne in 1901. Some years later he became the resident medical tutor at his college, and he retained his interest in and affection for it throughout his life. He graduated M.B., B.S. with honours in 1906.

In 1907-1908 he was resident medical officer and medical registrar at the Melbourne Hospital, and during this period

he collaborated with the late Sir Hugh Devine in the installation of the first comprehensive system of clinical case-recording to be adopted in Melbourne. In 1909 he was resident medical officer at the Children's Hospital, and in that year took his M.D. degree. In 1910 he went to London for post-graduate study. There he acted as clinical assistant to Sir James Mackenzie, and later he was associated with Sir Thomas Lewis in his study of digitalis and in his work on electrocardiography. He left London fully equipped as a physician and as a cardiologist, with the inestimable advantages of having worked in daily association with the two greatest cardiologists of their day. He brought back with him the first electrocardiograph to be used in medical practice in this country, and when the 1914-1918 war broke out he was recognized as a leader in the special field of cardiology. He retained this high position throughout his professional life.

Silberberg was working at the Melbourne Hospital in 1915 when he volunteered for service with the first Australian Imperial Force. It was characteristic of him that he sought no special status, and that his army career began as a general duties captain. He served as a junior physician in Egypt with the 14th Australian General Hospital; but in 1916 he contracted an obscure illness, was invalided back to Australia and remained seriously ill for three years. Then began the fruitful years of his life of service as teacher and consultant physician. In 1919 he was apopinted physician to out-patients to the Alfred Hospital, and in 1922 physician to the Children's Hospital and the Repatriation General Hospital, Caulfield. With the passage of time in 1925 he became physician to in-patients at the Alfred Hospital, Stewart Lecturer in Medicine at the University of Melbourne, and lecturer in medicine at the Dental School of the University of Melbourne. In 1938 he became a foundation Fellow of The Royal Australasian College of Physicians and later was elected to the Council. During the second World War he continued his work at the Alfred Hospital, from which he was due to retire in 1940. In 1945 he finally ceased his work there, and he became consultant physician not only to the Alfred Hospital but also to the Women's Hospital and the Children's Hospital.

It was my good fortune to become a resident medical officer at the Alfred Hospital at a time when Dr. Silberberg would have been in his middle forties. I cannot say that I had any close association with him at that time for I was house physician to Dr. Mackeddie, and when one was following some distance behind the wake of that colourful personality there was little time to stand and stare. Dr. Mackeddie was wont to say that the needle was the physician's only weapon, and in truth there were few places where a space could be found that we did not introduce it. It may be a sobering thought to the present generation to realize that they were forestalled so long ago. It is a sobering thought to me that as a young adult I could, on my daily rounds, see Dr. Silberberg about his duties in the medical wards and fail to see anything more than a quiet and, as I thought, austere individual with whom it would be difficult to find any common bond. Fate gave me a second chance, and I count it my great good fortune that after a lapse of years I came to know him well and to understand the seeming incongruity by which he was everywhere known as "Bill". He was not austere; he was a quiet and gentle person who trod softly through this world. To say this is not to deny that he was a personality with strength of character and strength of mind. These qualities sprang, I am sure, from his upbringing, and he was armed with those virtues without which no qualities of intellect will make a great or even a good physician. I learnt from him that the physician has weapons more pointed to his purpose than needles, for to be with him and to dwell in that atmosphere of quiet and wisdom must have brought solace to many a troubled person.

He loved the practice of medicine, and his associations with his professional colleagues. Many of us owe him a debt for his generous help in consulting practice on our return from overseas. After the war he fostered and maintained the local cardiac society, and we often enjoyed the hospitality of his home for our evening meetings. This society later played an important part in the formation of the Australasian Cardiac Society. Though he would never take credit for such things, it must have been a source of quiet satisfaction to him to see the development of the Australasian Cardiac Society, of which he became the chairman in 1954. Through Dr. Silberberg, the Alfred Hospital has a link with Sir James Mackenzie and Sir Thomas Lewis, and in the eminence of its present work in cardiology, the hospital is doubtless mindful of this historical association and of what it owes to its first cardiologist.

Dr. Silberberg faced all the milestones as he went on his way, and though he loved his profession there were wayside things that could engage him. He played golf and then bowls; he changed his large house for something smaller; but he and his wife carried from one to the other the warmth of a home. His life was honest and composed, and it was his home and his family, his books, his friends and his faith that sustained him to the end of his days. He leaves a widow and two sons, one of whom is following in his father's footsteps, and to them the sympathy of his colleagues will go out.

DR. G. A. BIRNIE writes: Early in 1920 an emergency hospital was opened in the Exhibition Building, Melbourne, to cope with the influenza epidemic. As a resident medical officer, it was there I first met Montefiore David Silberberg—known to all his friends as "Bill"—who had been appointed as visiting physician, and a friendship began which lasted nearly 40 years.

After service abroad with the Australian Army Medical Corps, Bill had worked with Sir James Mackenzie, and on return to Melbourne had commenced practice in Collins Street as a cardiologist, in which field he was to attain much eminence. At about the same time he was appointed honorary physician to out-patients at the Alfred Hospital, and visiting physician to the Repatriation General Hospital, Caulfield. At the Alfred Hospital, where his capacity was soon recognized, he well and truly laid down the foundations of the department of cardiology, the work of which has been so well carried on by his successors.

He early established his ascendancy in cardiology, and in the course of time developed a very large consulting practice. He was the first to introduce the electrocardiograph to Melbourne, and this early machine was a formidable piece of apparatus, occupying about a third of the space in his consulting room. There were no skilled technicians in those days, and on many a night Bill spent long hours dealing with its idiosyncrasies. Only those associated with him in those days could have realized the immense amount of work he carried out. On many occasions, after a long day's work, he would start off on a long motor-car drive to a country consultation, arriving back in the small hours of the morning; but at 9 a.m. he would arrive at the Alfred or Caulfield Hospital, and carry on in his usual calm and efficient manner.

To his honorary work at the Alfred Hospital he grudged no effort, and never allowed any private work to interfere with it. With students he was deservedly popular, and no one could have demonstrated more clearly the work of a first-class physician. The unfailing courtesy and consideration which put patients quickly at their ease; the careful history-taking; the methodical and thorough clinical examination; the clear and concise discussion of the various problems presented; all were indelibly imprinted on their minds.

In consultation he was a delightful colleague, always cooperative and helpful, with a wide cultural knowledge extending far beyond the bounds of his specialty, in which he was indeed a master. To those aware of the immense amount of work he got through, Bill's consistently calm and unhurried manner was a source of wonder. He was never known to display irritation of manner or ill-temper, and it is not without interest that he said once that, as a student, he had read Osler's great lecture "Æquanimitas", and had endeavoured to model his life on it.

His home life and background were a reflection of all that is fine and true in his religion and deep faith. Those who were fortunate enough to enjoy the hospitality of Bill and his charming wife realized how happy was his home life, and at the end he could in truth have said with Osler:

> I have loved no darkness,
> Sophisticated no truth,
> Nursed no delusion,
> Allowed no fear.

DR. C. SANDFORD WOOD writes: It was my happy lot to meet Dr. M. D. Silberberg many years ago, at the Alfred Hospital, then in consultation in private practice. In those days I worshipped Dr. Silberberg from afar, awed at his reputation, knowledge and sound common sense. As the years went by, many opportunities presented themselves of being in his company, and I learnt to admire M. D.'s charm, tact, modesty and courtesy; he was one of those happy souls who never criticized humans, but rather spoke of their good traits.

Once, when four of us were at lunch, I was very worried, having inadvertently performed a Rubin's test on a woman early in pregnancy. The first two doctors waxed rather facetious at my expense, and were of little comfort. Dr. Silberberg—by this time "Bill"—said, with his characteristic soft touch: "I am sorry that you are so worried."

Happy in his company, we freely speculated on life's journey, the tragedies and compensations of advancing years, of life and death, and the hereafter. Dr. Silberberg honoured his God, served his King and country, and gave great, wise and gentle service to his patients.

In the privacy of his home, "Bill" found love and comfort with his wife and family. To Mrs. Silberberg and her sons we extend our sympathy.

Medical Matters in Parliament.

HOUSE OF REPRESENTATIVES.

THE following extracts from *Hansard* relate to the proceedings of the House of Representatives on September 24, 1959.

Tuberculosis.

MR. CLEAVER: I preface my question to the Minister for Health by referring with appreciation and, I believe, a well deserved measure of congratulation, to the Minister's recent announcement that in Australia the number of deaths due to tuberculosis fell from 2169 in 1948 to 538 last year. As a result of recent isolated criticism of mass X-ray schemes, is the Minister able to indicate the value of mass X-rays in relation to the outstanding achievement in the eradication of tuberculosis?

DR. DONALD CAMERON: I do not think it would be too much to say that practically the entire success of the anti-tuberculosis measures depend upon mass X-ray. Early diagnosis is the key to the whole structure.

The following extracts from *Hansard* relate to the proceedings of the House of Representatives on September 29, 1959.

Poliomyelitis.

MR. ALLAN FRASER: I desire to ask the Minister for Health whether clinical tests are proceeding of the Cox oral anti-poliomyelitis vaccine. The question is prompted by the poor response to the adult anti-poliomyelitis Salk vaccine campaign which has been seemingly due to the innate dislike of injections by healthy adult human beings. In view of the importance of encouraging immunization against poliomyelitis, is special attention given to laboratory and field tests of the new oral vaccine to determine whether it can be accepted as a safe and valuable ally in the campaign against poliomyelitis?

DR. DONALD CAMERON: A great deal of information about oral anti-poliomyelitis vaccines is available to us in this country from experiments which have been conducted abroad. In addition some investigation is being undertaken at the Commonwealth Serum Laboratories into this aspect of the immunization of people against poliomyelitis. With respect, I would say to the honorable gentleman that I do not think it is really the fear of pain which deters people from receiving the poliomyelitis injection. I think it is the feeling of security which pervades the country on a rather insecure basis. While we have achieved a very high level of immunity among the child population of Australia, there is a large number of susceptible adults in the community. I would urge that they take advantage of the present mass immunization campaign to become immunized. Our present intention is not to change to any other form of vaccine because we have, at present, a highly effective vaccine, which must of course be given by injection. Until we are completely satisfied that it could be satisfactorily replaced by an oral vaccine we would be very unwilling to replace it.

Statistics of Blind Persons.

MR. DALY asked the Minister for Health, upon notice:

1. How many blind persons are there in (*a*) each State and (*b*) Australia, and in each category how many are (i) male and (ii) female?

2. How many (*a*) male and (*b*) female blind persons are in each age group?

3. How many blind persons in Australia are (*a*) married and (*b*) single?

4. How many blind persons in (*a*) each State and (*b*) Australia are living or maintained in homes for the blind?

DR. DONALD CAMERON: The answers to the honorable member's questions are as follows:

1. The only figures available are those of blind persons receiving age or invalid pensions. These figures exclude aliens and persons under the age of sixteen years, but represent fairly closely the number of all other blind persons resident in Australia. The following information regarding the number of blind persons who were receiving age or invalid pensions as at 30th June, 1959, has been furnished by the Department of Social Services:

(*a*)	New South Wales	1577
	Victoria	1479
	Queensland	685
	South Australia	452
	Western Australia	613
	Tasmania	274
	Northern Territory	5
	Australian Capital Territory	3
(*b*)	Australian total	5088

The most recent available classification into the number of males and females is in respect of blind persons receiving age or invalid pensions as at 30th June, 1957:

(i)	Males:	
	New South Wales (including Australian Capital Territory)	761
	Victoria	796
	Queensland	385
	South Australia (including Northern Territory)	166
	Western Australia	320
	Tasmania	115
	Australian total	2543
(ii)	Females:	
	New South Wales (including Australian Capital Territory)	613
	Victoria	674

Queensland	330
South Australia (including Northern Territory)	144
Western Australia	195
Tasmania	133
Australian total	2089

2. No classification into age groups is available.

3. The most recent available classification as to marital status is in respect of blind persons receiving age or invalid pensions as at 30th June, 1957:

(a) Married	2183
(b) Single	1575
Widowed, etc.	874

4. (a) Present figures:

New South Wales	55
Victoria	236
Queensland	16
South Australia	24
Western Australia	75
Tasmania	Nil
Australian Capital Territory	Nil
Northern Territory	Nil
(b) Australian total	406

The following extracts from *Hansard* relate to the proceedings of the House of Representatives on September 30, 1959.

Fluoridation of Water.

MR. BURY: My question to the Minister for Health concerns the fluoridation of public water supplies with a view to minimizing the decay of teeth, particularly in children. Is the Minister aware that a considerable number of very large cities in the United States already add fluorine to the public water supply for this purpose? Is he aware also that the research advisory committee of the National Health and Medical Research Council, at its 36th session in December, 1953, resolved that having considered published reports of the claims for and against artificial fluoridation of public water supplies, it recommended that, when the optimal intake is not obtained from natural sources, fluorine should be added to the public water supply? Have any steps been taken to add fluorine to the water supply of the Australian Capital Territory. If not, will the Minister look into the matter and consider whether the Commonwealth Government should set an example to State departments of health and water supply authorities by carrying out the recommendation of the council?

DR. DONALD CAMERON: I am aware of the various factors to which the honorable member has referred. The fluoridation of the Canberra water supply is not a simple matter because, as the honorable gentleman will know, some construction works are at present in progress in relation to the water supply and it would be premature to consider the question of fluoridation at this time.

Another factor to be considered in relation to the fluoridation of the water supply in the Australian Capital Territory is that Queanbeyan is served by the same supply and, therefore, there would need to be some coordination between the Commonwealth Government and the Government of New South Wales.

MR. WHITLAM: Queanbeyan wants to become part of the Territory.

DR. DONALD CAMERON: I do not know whether the fluoridation of the water supply will become an inducement or not. The matter will be considered in due course and in the light of the wishes of the inhabitants of the Territory.

SENATE.

THE following extracts from *Hansard* relate to the proceedings of the Senate on October 7, 1959.

Woollen Blankets.

SENATOR ROBERTSON: On 16th September I directed a question at the Minister for National Development. I understand that he now has a more detailed reply. The question was as follows:

I direct a question to the Minister representing the Minister for External Affairs, who is in charge of the Commonwealth Scientific and Industrial Research Organization. In explanation, I point out that during a recent lecture to the Institute of Public Supplies Officers, Mr. J. C. Dickinson, an officer of the International Wool Secretariat, said that statements being circulated regarding the unsuitability of woollen blankets for use in hospitals because of the excessive amount of wool fluff that resulted from their use, were not correct. Can the Minister say whether the C.S.I.R.O. has conducted research in this matter, and, if so, whether the results of that research can be made available to the Senate?

SENATOR SPOONER: The answer I have obtained from the Commonwealth Scientific and Industrial Research Organization is as follows:

Because of suggestions that fluff from wool blankets has been a significant source of cross-infection in hospitals the Division of Protein Chemistry of the C.S.I.R.O. has undertaken research into the composition of fluff collected in the wards of several hospitals in which wool blankets were used. The C.S.I.R.O. tests have shown fibres from blankets, or from any other source, constitute only a very minor fraction of the air-borne fluff. From the point of view of the welfare of the hospital patient the important consideration is the nature of the air-borne fluff at bed level. A typical sample of such fluff collected in the tests was found to contain 96 per cent of cotton and other forms of cellulose and only 3 per cent of wool. Up till the present time no comparative tests have been carried out in hospital wards where blankets made from textile fibres other than wool are used; however such tests are to be commenced in the near future.

Tests conducted by the Royal Melbourne Hospital in conjunction with C.S.I.R.O. have shown that, if the proper procedure is followed, shrink resistant all-wool blankets can be sterilized by boiling without deterioration. Blankets that have been subjected to more than 200 cycles of washing in the hospital laundry, involving boiling followed by tumble drying, still remain serviceable. The C.S.I.R.O. research has provided evidence that wool blankets can be used and laundered in hospitals very satisfactorily, avoiding the difficulties of both shrinkage and bacterial contamination. It has been shown that their use does not give rise to the formation of excessive amounts of fluff.

The following extracts from *Hansard* relate to the proceedings of the Senate on October 8, 1959.

Hospital Benefits Scheme.

SENATOR COOKE asked the Minister representing the Minister for Health, upon notice:

1. Is it a fact that many hospitals are being financially embarrassed because some patients, not covered by the Government's health scheme, are unable to pay for medical treatment and hospitalization, thereby leaving large amounts outstanding in the hospital accounts?

2. Is it a fact that in many cases it is almost impossible to recover outstanding amounts for treatment and accommodation, and that hospitals, particularly those outside metropolitan areas, are finding considerable difficulty in carying on economically?

3. Is it a fact that these amounts when written off or not collected, plus the expense occasioned in endeavouring to recover the outstanding moneys, are forcing up the costs of hospitalization for paying patients?

4. Will the Minister have an investigation made to ascertain whether a suitable system can be evolved to obviate this deficiency in the Australian health services scheme, and inform the Senate of the result of the investigation?

SENATOR HENTY: The Minister for Health has now furnished the following replies:

1, 2 and 3. As the provision and administration of hospitals is a State responsibility, I am not aware of the extent to which the matters stated constitute a significant problem for hospitals. Some published reports on these matters contain an element of exaggeration. I can say, however, that any difficulties being experienced are not due to any deficiency in the Commonwealth hospital benefits scheme, which, on the contrary, has gone far towards solving the problem of hospital finances.

4. In view of the foregoing, no.

Health and Medical Services.

SENATOR ARNOLD (through Senator Willesee) asked the Minister representing the Minister for Health, upon notice:

1. What is the total Commonwealth expenditure on the national health scheme?

2. What is the Commonwealth expenditure on hospital benefits?

3. What is the membership of registered organizations for hospital benefits?

4. What are the details of fund benefits and claims?

SENATOR HENTY: The Minister for Health has now furnished the following replies:

1. Commonwealth expenditure on the national health scheme was £59,578,993 for the year ended 30th June, 1959.

2. During the year ended 30th June, 1959, Commonwealth expenditure on hospital benefits was £14,802,290.

3. Membership of registered hospital benefits organizations at 30th June, 1959, was 2,749,308.

4. During the year ended 30th June, 1959, hospital fund benefit totalling £9,767,312 was paid for 725,627 claims.

Medical Benefits.

SENATOR ARNOLD (through Senator Willesee) asked the Minister representing the Minister for Health, upon notice:

1. What is the membership of registered organizations for medical benefits?

2. What are the particulars of claims for medical fund and medical fund benefits paid?

SENATOR HENTY: The Minister for Health has now furnished the following replies:

1. Membership of registered medical benefits organizations at 30th June, 1959, was 2,666,984.

2. During the year ended 30th June, 1959, medical fund benefit totalling £9,800,989 was paid for 16,383,164 professional services.

Pensioner Medical Service.

SENATOR ARNOLD (through Senator Willesee) asked the Minister representing the Minister for Health, upon notice:

1. What is the number of pensioners and dependants enrolled in pensioner medical services?

2. What is the payment to participating doctors?

3. What is the number of services and mileage claims?

SENATOR HENTY: The Minister for Health has now furnished the following replies:

1. As at 30th June, 1959, 720,043 pensioners and dependants were enrolled in the pensioner medical service.

2. During the year ended June 30, 1959, £3,806,457 was paid to participating doctors.

3. During the year ended June 30th, 1959, payment was made for 6,441,121 medical services and 43,241 mileage claims.

Commonwealth Serum Laboratories.

SENATOR ARNOLD (through Senator Willesee) asked the Minister representing the Minister for Health, upon notice:

In view of a letter which appeared recently in the "Australian Medical Journal" expressing doubt as to the value of anti-tetanic serum when used prophylactically, will the Minister advise the Senate as to the amount of anti-tetanic serum produced by the Commonwealth Serum Laboratories each year?

SENATOR HENTY: The Minister for Health has now furnished the following reply:

The information requested is as follows:

Production of anti-tetanus serum.

Year	Units
1956-57	728,000,000
1957-58	991,000,000
1958-59	863,000,000

The following extract from *Hansard* relates to the proceedings of the Senate on October 21, 1959.

Drugs.

SENATOR MCMANUS asked the Minister for Customs and Excise, upon notice:

1. Does the Department of Customs and Excise play any part in policing the ultimate disposal of narcotic drugs, after they have been imported into Australia?

2. What other Commonwealth departments play a part in ensuring that such drugs are disposed of legitimately?

3. How does Australia cooperate with the United Nations Organization, which seeks effective international control of narcotic drugs injurious to health?

SENATOR HENTY: I now furnish the following answers to the honorable senator's questions:

1. The powers of the Department of Custom and Excise over the control of narcotic drugs derive from the Customs Act, under which importers of narcotic drugs are required to be licensed. Under the terms of the licence, an importer may not dispose of narcotic drugs without the approval of the Collector of Customs. It is by this means that the Department of Customs and Excise is able to ensure that narcotic drugs in the possession of importers are disposed of only to persons or firms authorized under State legislation to be in possession of such drugs. The records kept by Australian wholesalers, retail chemists, hospitals etc., and their stocks of narcotic drugs, are policed by the relevant State government authorities. Each fraction of an ounce of any narcotic drug in the country must be accounted for.

2. Important aspects of the control of narcotic drugs are considered by the Department of Customs and Excise in consultation with the Commonwealth Department of Health and, through that department, with the Public Health Committee of the National Health and Medical Research Council. An inter-departmental committee, consisting of officers of the Departments of Health and Customs, on narcotic control, has been set up for this purpose. The Department of Territories is responsible for control of narcotic drugs in Australia's external territories.

3. Australia co-operates actively with the United Nations organization on the control of narcotic drugs, As a party to six international drug conventions and protocols, Australia has undertaken to apply stringent controls over the legitimate trade in narcotic drugs, so as to prevent possible diversions into the illicit traffic. The department also maintains a ceaseless campaign against the illicit traffic.

In so far as the legitimate trade is concerned, control is based on physical checks and records of all drug transactions required to be kept by licensed importers, wholesalers, etc., of narcotic drugs. Statistical information prepared from these sources is supplied to the United Nations, which receives similar material from all other parties to the Drug Conventions. A study of this information by the United Nations Drug Supervisory Body enables it to set world manufacturing levels for narcotics and to detect possible diversions of drug consignments into illicit traffic.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

PROGRAMME FOR 1960.

THE Post-Graduate Committee in Medicine in the University of Sydney announces the following programme of courses and activities for 1960.

Metropolitan and Week-end Courses.

The following metropolitan and week-end courses have been arranged: February 12, "Management of Cardiac Arrest" (limited to 10 candidates; closing date for applications February 1); February 20-21, week-end course in "Art of Teaching" (restricted to full-time and part-time lecturers in the Faculty of Medicine); March 12-13, week-end course in neurology; March 19-20, week-end course in electrocardiography; April 2-3, week-end course in thoracic diseases; April 6, 13, 20, 27, evening seminars, "Ischæmic Heart Disease"; May 9-20, general revision course; June 4-5, week-end course in rheumatic diseases; June 18-19, week-end course in renal diseases; June 25-26, week-end course in medicine, Rachel Forster Hospital for Women and Children; July 6, 13, 20, 27, evening seminars, "Cardio-vascular Diseases" (details yet to be announced); July 16-17, week-end course in endocrinology; July 23-24, week-end course in orthopædics; July 30-31, week-end

course in pædiatrics; August 6-7, week-end course in psychosomatic medicine; August 29-September 2, course in gynæcology and obstetrics at The Women's Hospital, Crown Street; August 29-September 9, "Reunion Week",[1] Royal North Shore Hospital; September 3-4, week-end course in gastro-intestinal diseases; September 10-11, week-end course in "Recent Advances in Cardio-vascular Diseases"; September, "Reunion Week",[1] St. Vincent's Hospital, and Old Sydney Hospitallers' Week;[1] September 24-25, week-end course in mental deficiency; October, "Reunion Week",[1] Royal Prince Alfred Hospital; October 5, 12, 19, 26, evening seminars, "Cardio-vascular Diseases" (details yet to be announced); October 8-9, week-end course in surgery, Rachel Forster Hospital; October, advanced course in dermatology; October-November, advanced course in surgery (see announcement below); November 14-25, anæsthetics course, Sydney Hospital (limited).

Arrangements are in hand to include courses in clinical respiratory physiology (conducted by the Department of Medicine), industrial medicine, and a group of three evening lectures, on a subject yet to be decided, at the Mater Misericordiæ Hospital, North Sydney.

An advanced course in surgery will take place some time in October or November, after the eighth Congress of the Pan-Pacific Surgical Association in Honolulu. It is expected that the following overseas lecturers will participate: Dr. Robert Linton, Boston; Dr. Ross Robertson, Vancouver; Dr. Henry Hawkins, Seattle; Dr. J. R. Heller, Director, Cancer Institute, Washington; Dr. George Pack, New York; Dr. Alton Oschner, New Orleans.

Electrocardiography Correspondence Course.

Negotiations are currently proceeding with the School of Medicine in the University of Southern California for the establishment by the Committee of a correspondence course in electrocardiography.

Resident and External Training Facilities.

Anæsthetics.

A limited number of post-graduate residencies is available for practical training in modern methods of anæsthetics at The Royal Newcastle Hospital. Fees, including board and residence, are £5 15s. 6d. per week of five days, Monday to Friday. A limited number of post-graduate residencies is also available at the Lewisham Hospital for a minimum period of three weeks. Fees, including board and residence, are £6 6s. per week of seven days. Lewisham Hospital reserves the right to select candidates for these residencies.

A full-time course in anæsthetics, non-resident, will be conducted by the Department of Anæsthetics and Resuscitation, Sydney Hospital, for two weeks from November 14. This course is limited to four applicants, the closing date for applications being June 30, 1960.

Blood Grouping and Transfusion.

On application to the Committee, instruction in the technique of blood grouping and transfusion can be arranged, free of charge, at the Red Cross Blood Transfusion Service or at The Royal Newcastle Hospital.

Gynæcology and Obstetrics.

Post-graduate residencies are available in gynæcology and obstetrics at The Women's Hospital, Crown Street, and at the Royal Hospital for Women, Paddington. Fees, including board and residence, are £5 15s. 6d. per week of seven days, and early enrolment with the Committee is essential.

External Studies.

Arrangements can be made with due notice to meet the individual needs of medical practitioners requiring à-la-carte courses. Fees are £1 1s. enrolment and £1 1s. per week.

General Residencies.

Post-graduate residencies are available for women graduates at the Rachel Forster Hospital for Women and Children, Redfern. Fees, including board and residence, are £6 6s. per week of seven days.

Post-Mortem Examinations.

On application to the Committee, instruction in the technique of the performance of post-mortem examinations can be arranged free of charge.

[1] Courses held in conjunction with other bodies (date yet to be confirmed).

Week-end Conferences, Country Centres.

The following dates and centres are all subject to confirmation: April 23-24, Bathurst; April 30-May 1, Wagga Wagga; April 30-May 1, Newcastle (gynæcology and obstetrics); June 11-12, Broken Hill; June 18-19, Katoomba; July 2-3, Hornsby; July 16-17, Wollongong; August 13-14, Newcastle (medicine); September 17-18, Armidale; September 24-25, Taree; September 24-25, Penrith; October 22-23, Albury; October 22-23, Newcastle (surgery); October 22-23, Parramatta; November 12-13, Bega-Cooma.

Diploma Courses.

The following diploma courses will be held: January 11, advanced medicine course begins (11 weeks); March 14, Final F.R.A.C.S. course[2] begins (six weeks, closing date February 12); March 14, D.D.R. I course begins (four months), D.T.R. I course begins (four months); May 9, D.D.M. I course begins (12 weeks), Primary F.R.A.C.S. course[2] begins (12 weeks, closing date March 4); June 6, D.P.M. I course begins (eight months); July 4, D.A. II course begins (three months), D.C.P. Group I course begins (seven months); July 18, D.P.M. II course begins (seven months), D.D.R. II course begins (eight months), D.T.R. II course begins (eight months); August 8, D.D.M. II course begins (seven months); August 29, D.A. I course begins (11 weeks), D.G.O. I course begins (11 weeks), D.O. I course begins (11 weeks), D.L.O. I course begins (11 weeks); September 19, D.C.P. Group II course begins (six months), D.C.P. Group III course begins (six months); November 14, D.G.O. II course begins (three months), D.L.O. II course begins (three months), D.O. II course begins (three months).

Annual Subscription Course.

The annual subscription course covers attendance at lectures by overseas lecturers and other specially arranged activities. The annual fee is £3 3s., from July 1, 1960. The fee for first and second year resident medical officers is £1 11s. 6d. A detailed diary card is printed at regular intervals throughout the year and posted to members. Occasionally last-minute alterations to meetings are necessary, and these are notified by advertisement in *The Sydney Morning Herald* ("Public Notices"), if possible on the day before the meeting.

Bulletin.

The annual subscription to the monthly *Bulletin of the Post-Graduate Committee in Medicine in the University of Sydney*, which consists of lectures given in the annual subscription course and other courses, has been increased to £2 2s. owing to increased printing, postal and other costs.

Film-Lending Services.

The Committee's film collection is housed and handled by the New South Wales Film Council, 55 Market Street. Borrowers must first be registered with the Committee, after which they may apply direct to the Council for individual films. A minimum of 10 working days' notice is required, and borrowers must bear the cost of dispatch and return of all films. A loose-leaf catalogue containing details and appraisals of medical films available for loan may be purchased from the Committee at a cost of £2 2s.

Lecture Recordings.

The Committee maintains a collection of recorded lectures, which it makes available for loan, free of charge, to medical practitioners and groups. These lectures are recorded on acetate discs, which may be played on standard microgroove equipment. In many instances they are illustrated with slides or photographs of slides.

Tape Collection.

Through the courtesy of E. R. Squibb and Sons, the Committee has established a collection of tape-recorded lectures especially directed towards the general practitioner. These recordings have been made by the Audio Digest Foundation of Glendale, California, and are now available for loan free of all charges except postage.

Overseas Bureau.

The Committee maintains an Overseas Bureau to assist medical practitioners proceeding abroad. A fee of £2 2s.

[2] Arranged by the New South Wales State Committee of the Royal Australasian College of Surgeons, in conjunction with the Department of Surgery in the University of Sydney.

[3] Arranged by the New South Wales State Committee of the Royal Australasian College of Surgeons.

is made for this service. Advice can be given concerning courses, accommodation, qualification for registration and other matters.

Taxation Deductions.

A deduction may be claimed in respect of fees paid for attendance at general practitioner courses held under the Committee's auspices when such fees are paid by practitioners who are in practice. Travelling expenses incurred in attending such courses may also be claimed as a deduction. When claims are made, it will be necessary to quote "Taxation File No. AF/1865".

General.

Dates of all courses should be confirmed, and further details regarding fees, diploma regulations and other information may be obtained on application to the Course Secretary, the Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

ROYAL PRINCE ALFRED HOSPITAL: EAR, NOSE AND THROAT DEPARTMENT.

Seminar Programme, 1959.

THE staff of the ear, nose and throat department of the Royal Prince Alfred Hospital will conduct a seminar on the second Saturday of every month at 8 a.m. in the Scot Skirving Lecture Theatre. The main speaker will not exceed forty minutes, and there will be a discussion at the conclusion of his remarks. All medical practitioners and clinical students are invited to attend.

At the next seminar, to be held on December 12, 1959, *Dr. G. C. Halliday will speak on "Footplate Techniques* in Mobilization of the Stapes".

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 58, of September 24, 1959.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

William O'Neill Shields (0310779) is appointed to a short-service commission, on probation for a period of twelve months, 24th June, 1959, with the rank of Flight Lieutenant.

The short-service commission of Squadron Leader A. V. Coleman (033029) is extended to 22nd September, 1960.

The due date for retirement of Squadron Leader A. V. *Coleman (033029) is extended to 22nd September, 1960.*

Active Citizen Air Force.

Medical Branch.

No. 25 (City of Perth) *Squadron.*—Flight Lieutenant J. A. Harley (034800) is transferred from the Reserve, 31st *January, 1959.*

Flight Lieutenant A. G. Fisher (051393) is transferred to the Reserve, 31st January, 1959.

Air Force Reserve.

Medical Branch.

Air Cadet Ian Anthony Musgrave (0115703) is provisionally appointed to a commission, 5th July, 1959, with *the rank of Pilot Officer.*

The following appointments, changes etc. are published in the *Commonwealth of Australia Gazette*, No. 60, of October 1, 1959.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointments.—Robert Charles Mulholland is appointed Surgeon Lieutenant (for Short Service) (on probation), *dated 2nd July, 1959.*

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Appointments.—Lieutenant Malcolm John Inglis, Royal Australian Naval Volunteer Reserve, is appointed Surgeon Lieutenant, with seniority in rank of 4th February, 1953, *dated 1st July, 1959.*

Promotions.—Surgeon Lieutenant Willoughby Sidney Sewell is promoted to the rank of Surgeon Lieutenant-Commander, dated 15th June, 1959.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/46827 Honorary Captain R. C. Rogers is appointed from the Reserve of Officers, and to be Captain (provisionally), 30th June, 1959. The provisional appointment of 1/59814 Captain B. E. Todd is terminated, 3rd September, 1959. To be *Captain (provisionally), 4th September, 1959*—1/59814 Bryan Edmund Todd.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/127895 Captain (provisionally) G. A. O'Neill relinquishes the provisional rank of Captain, 20th July, 1958, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command) and is granted the honorary rank of Captain, 21st July, 1958. 2/62339 Captain (provisionally) B. R. Hanley relinquishes the provisional rank of Captain, 28th June, 1959, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), and is granted the honorary rank of Captain, *29th June, 1959.* The resignation of 2/127073 Captain (provisionally) B. F. W. Quinn of his commission is accepted, 9th July, 1959. *To be Captains (provisionally), 12th August, 1959*—*2/130132* Roy Farley Thew and 2/130131 Geoffrey Burstal Field.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/101047 Captain (provisionally) D. J. Burne is seconded whilst in the United Kingdom, 25th March, *1959.*

Central Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 4/31918 Captain C. S. Kneebone is confirmed.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Southern Command.—Honorary Major R. H. Hadley and Honorary Captain H. B. O. Jones are retired, 31st August, 1959.

The following officers are placed upon the Retired List (Southern Command) with permission to retain their rank and wear the prescribed uniform, 31st August, 1959.

Major F. P. Donoghue and Captains E. J. Grieve and W. R. Rigg.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The dates on the notification regarding the grant of special leave without pay to Pilot Officer L. G. Trappett *(015935) as approved in Executive Council Minute No. 20* of 1959, appearing in Gazette No. 47 dated 30th July, 1959, are amended to read, 13th March, 1958, to 15th February, 1959, inclusive.

Active Citizen Air Force.

Medical Branch.

No. 22 (City of Sydney) Squadron.—Flight Lieutenant B. L. H. Leigh (0211247) is transferred to the Reserve, 3rd March, 1959.

Air Force Reserve.

Medical Branch.

The following former officer is appointed to a commission with the rank of Squadron Leader, 16th May, 1959:—R. P. V. Checcucci (0311661).

The following air cadets are provisionally appointed to a commission, 8th September, 1959, with the rank of Pilot Officer:—Andrew Barham Black (043049), Robert Barham *Black (043070), Ian Holden Buttfield (043086), Robert Mur*ray Edwards (043097), Anthony John Corin Holland (043105), Neville John Howard (043124), David Gilbert Jose *(043125), Creston Ivan Magasdi (043186).*

World Medical Association.

THIRTEENTH GENERAL ASSEMBLY.

THE Thirteenth General Assembly of the World Medical Association preceded by the thirty-sixth and followed by the thirty-seventh Council Sessions was held in Montreal, Canada, from September 6 to 12, 1959.

Dr. Charles Jacobson, Denmark, President of The World Medical Association, convened the Assembly and welcomed the participants. Greetings from Canada were extended to His Royal Highness The Duke of Edinburgh, President of the Canadian Medical Association, by Dr. E. Kirk Lyon, Deputy to the President, and from the Honorable Arthur Leclerc, M.D., Minister of Health for Quebec, by Dr. Tremblay. In addition the Honorable J. Waldo Monteith, Minister for National Health and Welfare of Canada, and the Honorable Senator Sarto Fournier, Mayor of Montreal, welcomed the delegates.

Installation of President.

Dr. Renaud Lemieux, of Canada, was elected and installed as President of The World Medical Association for 1959-1960. A new installation ceremony was initiated in which Dr. L. R. Mallen (Australia), Chairman of Council, paid tribute to Dr. Charles Jacobson, the retiring President, for the fine way in which he had carried out the duties of his offices, introduced the newly elected President to the assembly and administered a simple affirmation which Dr. Lemieux repeated after the Chairman of Council.

Keynote Address.

Dr. Norman H. Gosse, Canada, delivered the Keynote Address. His topic was "Our Obligations to Medicine and to International Goodwill".

New Members.

Applications for membership were received, and the following medical associations were approved for membership in The World Medical Association: The National Medical Union of Costa Rica (Dr. Jorge Salas Cordero, Apartado 548, San Jose, Costa Rica, C.A.); The Ghana Medical Association (Dr. F. T. Sai, Box 1596, Accra, Ghana).

Policies and Resolutions Adopted.

The Council was instructed to investigate the future objects and administrative affairs of the Association.

By-law 25 was amended to read (words in italics were added):

> 25. The discussions at the meetings of the General Assembly shall be conducted in the three official languages, that is, English, French and Spanish, and also in German, each delegate speaking in the language of his choice. It shall be the duty of the Council to arrange for interpretation at each meeting.

A similar amendment was made in Article 26 of the Standing Orders.

Regional Secretaries.

The Council in response to requests from member associations altered the assignment of member associations to regions in some instances which are noted below: Australia and Indonesia: Dr. J. G. Hunter (Australia). North Pacific (to include China, Korea, Japan, Philippines, Viet-Nam): (The Japan Medical Association to nominate the Regional Secretary). Europe, Morocco and Israel: Dr. M. Poumailloux (France). Asian area (to include Burma, India, Egypt, Pakistan, Thailand): Dr. Munawar Ali, Pakistan, in collaboration with Dr. A. P. Mittra, Council member from India. Latin America: Dr. Hector Rodriguez H. (Chile).

Corresponding Secretaries.

The following are corresponding secretaries. Union of South Africa: Dr. A. H. Tonkin. Ghana, Sudan: Secretaries of each Association to correspond direct with Secretary-General.

Fourteenth General Assembly.

The fourteenth General Assembly of the World Medical Association is scheduled to meet in Berlin, Germany, from September 15 to 20, 1960.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 31, 1959.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	2(1)	6(5)	..	..	..	..	..	8
Amoebiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	1	..	2(1)	..	..	..	10	..	13
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	5(5)	0(6)	..	..	..	..	1	4	10
Diphtheria	..	1(1)	..	2(2)	..	..	..	..	3
Dysentery (Bacillary)	..	1(1)	..	..	2(2)	..	..	..	3
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	90(36)	50(46)	55(5)	20(19)	3(3)	2(2)	..	..	220
Lead Poisoning	..	..	..	..	1	..	4	..	5
Leprosy	..	..	..	..	..	..	..	..	2
Leptospirosis	..	..	2	..	..	..	..	..	..
Malaria	..	1(1)	2(1)	..	1(1)	..	..	..	4
Meningococcal Infection	..	1(1)	..	..	..	..	..	..	1
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	..	..	..	..	..	..	..	1
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	15(11)	..	2	1(1)	..	..	2	20
Salmonella Infection	..	..	..	1(1)	..	..	..	..	1
Scarlet Fever	5(2)	8(5)	3	6(4)	2(1)	..	..	..	24
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	2(2)	..	51	..	53
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	18(13)	16(7)	10(6)	..	11(6)	2(1)	..	..	57
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] *Figures in parentheses are those for the metropolitan area.*

University Intelligence.

THE UNIVERSITY OF SYDNEY.

Result of Senate Elections.

THE quinquennial election of ten Fellows of the Senate of the University of Sydney by the graduates took place on Thursday, November 12, 1959. The formal votes recorded for each candidate were as follows:

Lieutenant-Colonel Sir Charles Bickerton Blackburn	3905
Mr. H. D. Black	2503
Mr. W. A. Selle	2391
Emeritus Professor F. A. Bland	2377
Major-General I. N. Dougherty	2373
Miss H. E. Archdale	2341
Mr. L. C. Robson	2229
Rev. B. R. Wyllie	2181
Professor A. K. Stout	2123
Emeritus Professor A. P. Elkin	2056

Dr. F. Lions	1957
Dr. C. G. McDonald	1956
Sir William Morrow	1541
The Honourable Mr. Justice J. H. McClemens	1472
The Honourable Mr. Justice C. McLelland	1453
Dr. W. P. MacCallum	1438
Dr. A. J. Murray	1321
Mr. H. F. Benning	1274
Mrs. L. Hayter	1175
Dr. D. J. Anderson	1151
Mr. J. M. Gosper	1134
The Honourable Mr. Justice R. Else-Mitchell	1122
Dr. J. G. Hunter	1117
Mr. C. L. Devenish-Meares	994
Associate Professor J. F. D. Wood	927
Dr. H. Selle	877
Dr. J. G. Watson	838
Mr. W. E. R. Francis	779
Miss J. R. Delaney	760
Dr. L. E. McDermott	645
Dr. G. L. Howe	617
Dr. R. E. Buckingham	549
Mr. C. R. Storey	533
Mr. M. D. Broun	400
Mr. G. N. Bowles	231

Medical Appointments.

Dr. C. J. Cooper, Dr. J. I. Manson and Dr. P. Hoopman have been appointed Senior Resident Medical Officers at the Royal Adelaide Hospital, Adelaide.

Dr. B. F. W. Quinn, Dr. J. M. B. Wane, Dr. G. J. B. Robinson and Dr. E. A. Freeman have been appointed Senior Resident Medical Officers at the Royal Adelaide Hospital, Adelaide.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Ballenden, John St. Clair, M.B., Ch.B., 1941 (Univ. Witwatersrand), D.M.R.D., R.C.P. and S., 1953, Suite 2, Tome's Buildings, Railway Street, Rockdale, New South Wales.

Deaths.

THE following deaths have been announced:

PARKER.—Leslie Richard Parker, on November 20, 1959, at Five Dock, New South Wales.

MACCALLUM.—Walter Paton MacCallum, on November 22, 1959, at Sydney.

HARPUR.—Malcolm Denholme Harpur, on November 23, 1959, at Woolwich, New South Wales.

HOLMES.—Horace Iles Holmes, on November 24, 1959, at Warrnambool, Victoria.

Diary for the Month.

DECEMBER 7.—Victorian Branch, B.M.A.: Executive of Branch Council.
DECEMBER 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
DECEMBER 9.—Victorian Branch, B.M.A.: Branch Council.
DECEMBER 10.—New South Wales Branch, B.M.A.: Branch Meeting.
DECEMBER 11.—Tasmanian Branch, B.M.A.: Branch Council.
DECEMBER 11.—Queensland Branch, B.M.A.: Council Meeting.
DECEMBER 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DECEMBER 17.—New South Wales Branch, B.M.A.: Public Relations Committee.
DECEMBER 22.—New South Wales Branch, B.M.A.: Hospitals Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £6 *per annum* within Australia and the British Commonwealth of Nations, and £7 10s. *per annum* within America and foreign countries, payable in advance.